# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

Filed: July 16, 2025

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| TIM A. ZIKELI, | * | PUBLISHED |
| Petitioner, | * | No. 20-564V |
| v. | * | Special Master Nora Beth Dorsey |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | Dismissal; Influenza ("Flu") Vaccine; Sudden Sensorineural Hearing Loss ("SSNHL"). |
| Respondent. | * | |

Ronald Craig Homer, Conway, Homer, P.C., Boston, MA, for Petitioner.
Alexa Roggenkamp, U.S. Department of Justice, Washington, DC, for Respondent.

## DECISION[1]

## I. INTRODUCTION

On May 6, 2020, Tim A. Zikeli ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that he suffered sudden sensorineural hearing loss ("SSNHL") as a result of an influenza ("flu") vaccine he received on October 23, 2018. Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating that "this case is not

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

appropriate for compensation under the terms of the [Vaccine] Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 23).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards, the undersigned finds that Petitioner failed to provide preponderant evidence that his flu vaccine caused his SSNHL. Thus, Petitioner has failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is not entitled to compensation.

## II. ISSUES TO BE DECIDED

The parties stipulate that Petitioner received a flu vaccination on October 23, 2018 in the United States. Joint Submission, filed Mar. 29, 2024, at 1 (ECF No. 108). The parties agree Petitioner was properly diagnosed with SSNHL. Id.

In dispute is causation, specifically all three Althen prongs: (1) whether the flu vaccine can cause SSNHL; (2) whether "there is a logical sequence of cause and effect between [Petitioner's] October 23, 2018 flu vaccination and the development of his SSNHL," and (3) whether "the onset of [Petitioner's] SSNHL occurred within a medically appropriate timeframe with respect to his October 23, 2018 flu vaccination." Joint Submission at 2.

## III. BACKGROUND

### A. Procedural History

Petitioner filed his petition on May 6, 2020, followed by medical records[3] in May and June 2020. Petition; Petitioner's Exhibits ("Pet. Exs.") 1-15. This case was assigned to the undersigned on July 21, 2020. Notice of Reassignment dated July 21, 2020 (ECF No. 17). Respondent filed his Rule 4(c) report, arguing against compensation, on December 18, 2020. Resp. Rept. at 1.

On June 9, 2021, Petitioner filed an expert report from Dr. Edwin Monsell. Pet. Ex. 16. Respondent filed an expert report from Dr. Herman F. Staats on January 5, 2022 and an expert report from Dr. Yu-Lan Mary Ying on February 7, 2022. Resp. Exs. A, C.

Thereafter, at request of the parties, the undersigned held a Rule 5 conference on April 14, 2022. Rule 5 Order dated Apr. 14, 2022 (ECF No. 58). The undersigned was unable to provide any preliminary findings as to causation. Id. at 2. Respondent indicated he wished to continue to defend this matter, and an entitlement hearing was scheduled pursuant to Petitioner's request. Resp. Status Rept., filed July 15, 2022 (ECF No. 67); Order dated Aug. 30, 2022 (ECF No. 70); Prehearing Order dated Sept. 28, 2022 (ECF No. 73).

Supplemental expert reports from Dr. Monsell, Dr. Staats, and Dr. Ying were filed by the parties in 2023. Pet. Ex. 63; Resp. Exs. E-F. In October 2023, the parties indicated they were

[3] Updated medical records were filed throughout litigation.

amenable to resolving this matter through a ruling on the record in lieu of an entitlement hearing. Joint Status Rept., filed Oct. 4, 2023 (ECF No. 93). The entitlement hearing was cancelled, and a briefing schedule was set. Id.; Order dated Jan. 22, 2024 (ECF No. 99).

Petitioner filed his motion for a ruling on the record on March 29, 2024. Pet. Motion for an Entitlement Ruling on the Record ("Pet. Mot."), filed Mar. 29, 2024 (ECF No. 110). Respondent filed his response on July 8, 2024, and Petitioner filed a reply on August 8, 2024. Resp. Response to Pet. Mot. ("Resp. Response"), filed July 7, 2024 (ECF No. 118); Pet. Reply to Resp. Response ("Pet. Reply"), filed Aug. 8, 2024 (ECF No. 119). Respondent filed a sur-reply on August 27, 2024. Resp. Sur-Reply to Pet. Mot. ("Resp. Sur-Reply"), filed Aug. 27, 2024 (ECF No. 122). Petitioner did not file a response.

This matter is now ripe for adjudication.

## B. Factual History

### 1. Relevant Medical History[4]

On September 11, 2018, Petitioner saw his primary care physician ("PCP"), Dr. Paul H. Zuzick, for an annual examination. Pet. Ex. 2 at 99. Petitioner denied any concerns or active problems. Id. During a review of systems, Petitioner denied hearing loss. Id. at 100. Petitioner was taking simvastatin (Zocor)[5] for high cholesterol,[6] which Dr. Zuzick recommended holding "to see if the medication [was] necessary." Id. at 99, 101.

On October 23, 2018, Petitioner received the flu vaccination at issue. Pet. Ex. 1 at 1.

[4] This summary of medical records is largely taken from the parties' briefs and Respondent's Rule 4(c) report, as the undersigned finds the parties provided an accurate representation of the records. See Pet. Mot. at 4-12; Resp. Response at 1-4; Resp. Rept. at 1-4. The undersigned has edited the summary.

[5] Simvastatin is "used to lower blood lipid levels in the treatment of hypercholesterolemia and other forms of dyslipidemia and to reduce the risk of morbidity and mortality associated with atherosclerosis and coronary heart disease." Simvastatin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=45888 (last visited June 27, 2025).

[6] Petitioner's medical records and the parties' experts refer to Petitioner's chronic high cholesterol as hypertriglyceridemia (elevated triglycerides in the blood) and hyperlipidemia (elevated lipids in the plasma). Hypertriglyceridemia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24052 (last visited June 27, 2025); Hyperlipidemia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=23870 (last visited June 27, 2025).

Three days later, on October 26, 2018, Petitioner presented to Nurse Practitioner ("NP") Kristin Kastner complaining of "Ear Problem" that "started [one] week ago."[7] Pet. Ex. 3 at 116-18. "Associated symptoms" included "clear rhinorrhea," which was described as chronic and associated with allergies. Id. at 118. He was taking daily oral allergy medication. Id. He tried over-the-counter hydrogen peroxide ear wash for symptom relief and he attributed his complaints to "ear wax." Id. Review of systems was positive for decreased hearing in the right ear. Id. Physical examination revealed no fever, no lymphadenopathy, slightly enlarged or swollen tonsils, normal external ears, clear ear canals, and clear tympanic membranes with all landmarks noted. Id. at 118-19. Assessment was obstruction of right eustachian tube. Id. at 119. NP Kastner noted Petitioner's symptoms "may be due to [a] small amount of middle/inner ear fluid due to chronic allergies." Id. There was no accumulation of wax noted. Id. She recommended treatment with pseudoephedrine[8] and fluticasone nasal spray and to follow up with his PCP if symptoms worsened or failed to improve. Id.

On October 30, 2018, Petitioner saw audiologist Janice Beaton and otolaryngologist Dr. Anders Holm. Pet. Ex. 2 at 103-16. Petitioner reported his right-sided hearing loss "began last Thursday"[9] with a duration of five days. Id. at 112. He denied a history of hearing loss or upper respiratory symptoms and reported no other symptoms. Id. at 104, 113. He had "some history of noise exposure from using power equipment around his home but [he] use[d] ear protection." Id. at 104. He denied tinnitus,[10] otitis media, otalgia, and dizziness. Id. Dr. Holm's physical examination of Petitioner's right ear was normal, and there was no redness or abnormality of the tympanic membrane. Id. at 114. Petitioner had "[n]ormal clinical speech reception threshold (whispered voice, finger rub)." Id.

Audiometric test results revealed moderate to severe sensorineural hearing loss ("SNHL") in the right ear with no word recognition abilities in the right ear. Pet. Ex. 2 at 104-05. In the left ear, there was "[n]ormal hearing" with a "mild to moderate [SNHL] above [1000 Hz]." Id. at 104. Audiologist Beaton's impression was "mild to moderate sensorineural loss" above 1000 Hz in the left ear and "moderate to severe [SNHL] in the right with no word recognition abnormalities in the right ear." Id. at 105. Dr. Holm assessed Petitioner with "[a]cute [SNHL] [in] the right ear" with "absence of speech discrimination with preserved hearing on the left-hand side." Id. at 115. Dr. Holm prescribed high-dose prednisone and recommended follow up in one week. Id.

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[7] One week prior would be October 19.

[8] Pseudoephedrine is "one of the stereoisomers of ephedrine, having less pressor action and fewer central stimulant effects than ephedrine." Pseudoephedrine, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=41684 (last visited June 27, 2025).

[9] "[L]ast Thursday" would be October 25, 2018.

[10] Tinnitus is "a noise in the ears, such as ringing, buzzing, roaring, or clicking. It is usually subjective in type." Tinnitus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=50114 (last visited June 27, 2025).

Petitioner followed up with audiologist Beaton and Dr. Holm on November 6, 2018, and he reported no improvement and worsening tinnitus. Pet. Ex. 2 at 118, 124. Repeat audiometric testing showed severe SNHL in the right ear with "some reduction in thresholds" compared to testing the prior week. Id. at 119. Dr. Holm treated Petitioner with a trans-tympanic steroid injection and ordered a magnetic resonance imaging ("MRI"). Id. at 124-25.

On November 9, 2018, Petitioner saw audiologist Kiz Robertson. Pet. Ex. 2 at 131. He reported right-sided SSNHL that began on October 25, 2018 with no change in hearing and continued tinnitus following treatment with prednisone and trans-tympanic steroid injection. Id. Repeat audiometric test revealed "[s]evere to profound mostly [SNHL] from 250 through 8000 Hz" in the right ear and "[n]ormal hearing [] from 250 through 1000 Hz with a borderline normal/mild to moderately severe hearing loss from 2000 through 8000 Hz" in the left ear." Id. Impression was "[h]earing loss in both ears with the right ear significantly worse . . . and a diagnosis of right [SSNHL] with restricted hearing in the contralateral ear." Id.

Petitioner called Dr. Holm's office on November 14, 2018 asking if "his flu shot [a] couple days before he started having his hearing loss . . . may have something to do with his issues." Pet. Ex. 3 at 161. A registered nurse ("RN") called Petitioner back and advised, "[p]er Dr. Holm, the flu shot would not cause current symptoms." Id.

Petitioner underwent a brain MRI on November 19, 2018. Pet. Ex. 4 at 16-17. No acute intracranial process was identified, and the radiologist noted the internal auditory canal was unremarkable. Id. at 17. Additionally, "[m]ild periventricular abnormal signal intensity, likely reflecting small vessel ischemic changes," was seen. Id.

On November 20, 2018, Petitioner requested a refill of prednisone from Dr. Holm because "he felt it helped with tinnitus" although he "[was] reporting no improvement in hearing." Pet. Ex. 2 at 150. Dr. Holm filled a new prednisone prescription and directed to follow up in one month. Id. That same day, Petitioner called Dr. Zuzick, requesting that he "be made aware of [Petitioner's] hearing loss and steps he's been taking." Id. at 153. On this call, Petitioner reported his hearing loss began on October 25, 2018. Id.

A Vaccine Adverse Event Reporting System ("VAERS") report was submitted by pharmacist Jonathan Luetke on Petitioner's behalf on November 20, 2018. Pet. Ex. 8 at 3. The report documented the date the adverse event started as October 25, 2018. Id. It stated that "[w]ithin 48 hours after receiving the [flu] vaccine, patient experienced a sudden loss of hearing in his right ear. He was taking a hike and listening to music with headphones in both ears. As he was driving home, he noticed the loss of hearing accompanied by tinnitus." Id. at 3-4.

On November 30, 2018, Petitioner saw audiologist Karen Belgard for consultation due to SSNHL that began on October 25, 2018. Pet. Ex. 5 at 13. She recommended further evaluation for hearing aids. Id.

Petitioner returned to audiologist Robertson on December 20, 2018 for continued hearing loss and tinnitus in his right ear. Pet. Ex. 2 at 165. Repeat audiometric testing impression

confirmed "[c]ontinued hearing loss with the right ear worse [than] the left ear and a diagnosis of right [SSNHL] and right tinnitus." Id. at 166. Petitioner also saw Dr. Holm on that day. Id. at 171. Dr. Holm documented Petitioner reported "some improvement but still ha[d] some degree of tinnitus and distortion of his hearing." Id. Dr. Holm noted "[a]udiology continues to reveal an improved pattern." Id.

On March 12, 2019, Petitioner presented to a Costco hearing center "for screening to see if his hearing [was] getting better from a flu shot provoked [hearing loss]." Pet. Ex. 9 at 11. The audiologist noted that his hearing "show[ed] marked improvement" from the last two tests done at other facilities. Id. The audiologist agreed Petitioner has moderate SNHL in his right ear and mild to moderate hearing loss, described as age-related, in the left ear. Id. Petitioner returned to Costco on April 25, 2019 for a hearing test, which showed worse pure tone average in right ear when compared to the March 2019 test. Id.

Petitioner was evaluated for hearing aids on May 16, 2019. Pet. Ex. 6 at 5. During the evaluation, he "reported concerns about the intensity of his tinnitus in the right ear." Id. Petitioner was fitted for hearing aids on May 29, 2019, and by June 13, 2019, Petitioner reported that he was hearing well with the hearing aids. Id. at 8, 11.

On October 31, 2019, Petitioner saw his PCP, Dr. Zuzick, for an annual examination. Pet. Ex. 12 at 36. Dr. Zuzick noted Petitioner "ha[d] a history of sudden hearing loss on the right side which may be related to receiving a [flu] vaccination."[11] Id. Dr. Zuzick did not make any notations regarding Petitioner's use of hearing aids or the condition of his hearing at the time of this visit. See id. at 36-39.

On February 21, 2020, Dr. Zuzick's office called Petitioner regarding his annual flu vaccine. Pet. Ex. 12 at 44-45. The note from this call stated,

> [Petitioner] did not get a flu shot this year. In Oct[ober] 2018 patient got the flu shot and [two] days later patient lost hearing in right ear. Patient states he discussed with doctor at last visit. Patient had testing done and none of the doctors attributed his hearing loss to the flu shot but the patient does.

Id. at 45.

Petitioner has continued to have his hearing aids checked approximately every six months. See Pet. Exs. 61-62.

[11] Dr. Zuzick's "Review of Systems" documented "[d]enies . . . hearing loss," which appears to be an error. Pet. Ex. 12 at 37.

### 2. Petitioner's Declaration[12]

Petitioner averred that he was in good health prior to the flu vaccination at issue other than checkups for high cholesterol. Pet. Ex. 13 at ¶ 1. Petitioner "often received an annual flu vaccine and never had adverse reactions." Id.

Petitioner reported he received a flu vaccination around 2:00 P.M. on October 23, 2018. Pet. Ex. 13 at ¶ 2. He "felt well that day." Id.

Two days later, on October 25, 2018, Petitioner "realized that [he] was unable to hear out of [his] right headphone" while hiking that afternoon. Pet. Ex. 13 at ¶ 3. He was listening to music with headphones on a hike when he suddenly had "complete hearing loss on [his] right side" while his hearing on the "left was unchanged and normal." Id. He stated he "never previously experienced anything like it." Id.

On October 26, 2018, three days post-vaccination, Petitioner presented to a medical provider who "examined both ears and confirmed there was no obstruction or fluid in the ears." Pet. Ex. 13 at ¶ 4. Petitioner took Sudafed and nasal spray, but after three days, he had "no relief or return of [his] hearing." Id.

One week post-vaccination, Petitioner had his first audiology appointment with audiologist Beaton and Dr. Holm and was diagnosed with SSNHL in his right ear. Pet. Ex. 13 at ¶ 5. Thereafter, Petitioner was treated with prednisone and an intratympanic steroid injection and he underwent an MRI. Id. at ¶¶ 5-6. Petitioner saw no improvement and his MRI was normal. Id. Petitioner returned to the pharmacy at which he received his flu vaccination, and a pharmacist completed a VAERS form. Id. at ¶ 7. Petitioner also sought a second opinion, underwent acupuncture, and received numerous audiograms and his hearing loss remained. Id. at ¶¶ 8-12.

Petitioner obtained hearing aids in 2019, which provided him with the ability to "pick up more sound;" "mask the tinnitus," although it was still present; and "engage with others better than [he] [could] without them." Pet. Ex. 13 at ¶ 12. He averred that he "participate[s] in conversations and interact[s] with [his] surroundings very differently than [he] did prior to the hearing loss." Id.

## C. Sudden Sensorineural Hearing Loss

SNHL occurs "when there is damage to the inner ear (cochlea) or to the nerve pathways from the inner ear to the brain." Pet. Ex. 22 at 1.[13] It is the most common type of permanent

[12] Although titled an affidavit, it was not notarized. Therefore, it is referenced as a declaration. Petitioner also submitted a declaration stating no prior civil action had been filed for his alleged vaccine injury. See Pet. Ex. 14.

[13] Am. Speech-Language-Hearing Ass'n, Type, Degree, and Configuration of Hearing Loss, Audiology Info. Series, 2015.

hearing loss. Id. This form of hearing loss "reduces the ability to hear faint sounds," and even if speech is "loud enough to hear, it may still be unclear or sound muffled." Id. Possible causes include toxic medication or drugs, genetic or hereditary, or aging. Id. SNHL is "generally idiopathic," but some cases are "associated with infections, vasculitides, tumors, [] genetic conditions, and cardiovascular risk factors." Pet. Ex. 24 at 1.[14]

SSNHL is a subset of SNHL that "occurs within a 72-hour window" and meets specific audiometric criteria. Pet. Ex. 26 at 3.[15] Idiopathic SSNHL has "no identifiable cause despite investigation" and accounts for "90% of patients with SSNHL." Id.

### D. Expert Reports and Medical Literature

#### 1. Petitioner's Expert, Dr. Edwin Monsell[16]

##### a. Background and Qualifications

Dr. Monsell is "board-certified by the American Board of Otolaryngology—Head and Neck Surgery and hold[s] a Certificate of Added Qualifications from the same board in Neurotology (diseases and surgery of the inner ear and related skull base)." Pet. Ex. 16 at 1; see also Pet. Ex. 109 at 4. He obtained a Ph.D. in cell biology and neuroscience from Duke University in 1977 and an M.D. from University of North Carolina School of Medicine in 1979. Pet. Ex. 109 at 1. He then completed a surgical internship and a residency in the Otolarynogology—Head and Neck Surgery department at Northwestern University, followed by a fellowship at the House Ear Institute in Los Angeles, CA. Id. Since 1986, Dr. Monsell has held various hospital or other professional appointments and since 2000, he has also held faculty appointments. Id. at 1-2. He currently holds two hospital appointments in Seattle, Washington and teaches in the Otolaryngology-Head and Neck Surgery department at University of Washington in Seattle. Id. at 1-2. He is a member and has held various positions for the American Academy of Otolaryngology—Head and Neck Surgery, Association for Research in Otolaryngology, and other professional societies. Id. at 2-3; Pet. Ex. 16 at 1. In 2003, Dr. Monsell "received the Harris P. Mosher Award, the highest award for clinical and translational research in Otolaryngology by the American Laryngological, Rhinological[,] and Otological Society (a/k/a the Triological Society) for [his] research on mechanisms of hearing loss." Pet. Ex. 16 at 1. Throughout his career, he has published on the mechanisms of hearing loss, "treated many thousands of patients with hearing loss[,] and performed over 3,500 major ear operations to remove tumors, infection[,] and restore hearing." Id.; see also Pet. Ex. 109 at 10-18.

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[14] Roger Baxter et al., Sudden-Onset Sensorineural Hearing Loss After Immunization: A Case-Centered Analysis, 155 Otolaryngology Head & Neck Surgery 81 (2016).

[15] Sujana S. Chandrasekhar et al., Clinical Practice Guideline: Sudden Hearing Loss (Update), 161 Otolaryngology Head & Neck Surgery S1 (2019).

[16] Dr. Monsell provided two expert reports. Pet. Exs. 16, 63.

**b. Opinion**

To provide context for his opinions, Dr. Monsell provided background information about the inner ear, describing it as "a collection of complex biological systems that function to encode the frequencies and intensities of sound into a neural code that is then transmitted to the auditory central nervous system." Pet. Ex. 63 at 1. This system is comprised of many small "vascular, immunologic, neural, and sensory structures surrounded by dense bone." Id. He explained the process by which vibrations of sound enter the ear canal and are coded within the inner ear, the blood supply, and the spiral ligament, noting that injury to any component can lead to SSNHL. Id. at 2.

In his first report, Dr. Monsell explained the cochlea (inner ear) is susceptible to ischemic injury (oxygen starvation) when the microvasculature (small blood vessels including arterioles and capillaries) is compromised. Pet. Ex. 16 at 14. He posited that the microvasculature of the inner ear is regulated by nitrous oxide pathways and that an upregulation of inflammatory cytokines and nitric oxide synthase[17] could lead to microvascular pathology. Id. at 15. Further, labyrinthitis (inner ear inflammation) can compromise ion homeostasis in the ear, affecting the microvasculature of the cochlear lateral wall and stria vascularis,[18] resulting in damage to hearing function. Id. Given this background, Dr. Monsell opined that the flu vaccine "can occasionally—but quite rarely—provoke a local inflammatory response of the inner ear" resulting in permanent damage. Id. at 14.

**i. Althen Prong One**

Although "[m]ultiple mechanisms are implicated in the process of SSNHL," Dr. Monsell acknowledged the exact mechanisms at play in SSNHL are "not completely understood." Pet. Ex. 16 at 13; Pet. Ex. 63 at 13. He proposed that the flu vaccine can cause SSNHL "based on idiosyncratic microvascular and inflammatory reactions." Pet. Ex. 16 at 17; see also Pet. Ex. 63 at 21.

In his first expert report, he very briefly summarized how a flu vaccine can cause SSNHL: following a flu vaccination, "local inner ear inflammatory responses [lead] to activation

[17] Synthase is "used in the trivial or recommended names of some enzymes . . . when the synthetic aspect of the reaction is dominant or emphasized." Synthase, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=48589 (last visited June 27, 2025).

[18] Stria vascularis ductus cochlearis (vascular stria of cochlear duct) is "a layer of vascular tissue consisting of epithelial cells, mesothelial cells, and probably some neuroectoderm" that "covers the outer wall of the cochlear duct and is thought to secrete the endolymph." Stria Vascularis Ductus Cochlearis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=108900 (last visited June 27, 2025).

of [nuclear factor kappa B ("NFKB" or "NFκB")[19]] pathways [that] result[] [in] a destructive response in the inner ear, leading to cell death and resultant unilateral severe deafness." Pet. Ex. 16 at 17.

In his second expert report, Dr. Monsell proposed a more-detailed mechanism: the "post-vaccination inflammatory response[] led to development of inflammation, reduced cochlear blood flow, injury/reperfusion of [the] right inner ear, creation of reactive oxygen species [("ROS")[20]], and activation of cellular signaling pathways and events, leading to cell death and resultant unilateral profound sensorineural deafness." Pet. Ex. 63 at 6-7, 21. According to Dr. Monsell, this detailed mechanism contains seven steps to explain how the flu vaccine can trigger SSNHL. Id. at 6-7.

Each step is set forth below, along with a brief analysis of Dr. Monsell's supportive medical literature. See Pet. Ex. 63 at 6-12.

**1. Seven-Step Mechanism and Supporting Medical Literature**

I. Flu vaccination stimulates the Acute Phase Reaction ("APR"),[21] which includes release of inflammatory cytokines into the circulation, fibrinogen, tumor necrosis factor ("TNF")[22], NFκB, and others.

Dr. Monsell stated "that systemic immune stimulation can lead to activation of NFκB in the spiral ligament of the cochlea, resulting in damage to important hearing structures and may interact with other components of the proposed mechanism." Pet. Ex. 63 at 20. "That [flu] vaccination could plausibly be such a stimulant or trigger in rare cases cannot be dismissed." Id.

[19] NFKB is "a transcription factor that plays an important role in the expression of proinflammatory genes that are associated with apoptosis, tumorigenesis, inflammation, arthritis, and some autoimmune conditions." Nuclear Factor κB, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74572 (last visited June 27, 2025).

[20] Reactive oxygen species, or ROS, are "biologically active, partially reduced derivatives of molecular oxygen ($O_2$), including the superoxide radical, hydrogen peroxide, and the hydroxyl radical. They are produced by normal metabolic processes and may also be produced by the absorption of energy, such as ultraviolet or ionizing radiation, and can damage biological systems." Reactive Oxygen Species, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=107488 (last visited June 27, 2025).

[21] For additional discussion by Dr. Monsell about APR, see Pet. Ex. 63 at 19-20.

[22] TNF refers to "either of two lymphokines that are capable of causing in vivo hemorrhagic necrosis of certain tumor cells but not affecting normal cells; they have been used as experimental anticancer agents but can also induce shock when bacterial endotoxins cause their release." Tumor Necrosis Factor, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74613 (last visited June 27, 2025).

In support, Dr. Monsell cited Khalil and Al-Humadi,[23] who discussed types of "acute phase reactants" and their potential importance relative to vaccination. Pet. Ex. 76 at 1. Acute phase reactants are defined as "a group of 11 key proteins," eight of which have positive effects and three with potentially undesired effects. Id. at 6. The authors posited that changes in the expression of these proteins vary due to individual genetics, age, or other factors, and may alter the response to any given vaccine. Id.

Khalil and Al-Humadi listed the acute phase reactants associated with vaccines. Pet. Ex. 76 at 5 tbl.II. The acute phase reactants C-reactive protein ("CRP")[24] and transthyretin[25] were identified as associated with the flu vaccine based on a study published in 2006. Id. (citing Pet. Ex. 70).[26] CRP was identified as a "[p]ositive acute phase reactant[]." Id. at 3, tbl.I. It is a "plasma protein . . . produced as a result of pro-inflammatory cytokine signaling" that is "elevated during infection or inflammation as part of the innate immune response."[27] Id. at 2. The authors suggested that CRP has potential value as a "diagnostic marker for active inflammation and infection." Id. Other than its designation as a diagnostic marker for inflammation, the authors did not identify any adverse effects of CRP after the flu vaccination.

Transthyretin (also known as prealbumin) "is a negative [acute phase reactant] synthesized and excreted by the kidneys and gastrointestinal tract." Pet. Ex. 76 at 5. The authors noted that it "functions as a biomarker for predicting poor short-term outcome and disease severity in patients with burn injuries or respiratory failure." Id. (internal citations omitted). Low levels have been associated with an "increase in mortality," and low preoperative levels are associated with "increased risk of postoperative infections." Id. Khalil and Al-Humadi did not identify any risk of SSNHL or other hearing loss due to CRP or transthyretin. The specific proteins listed by Dr. Monsell in his expert report, including fibrinogen, TNF, and NFKB, were

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[23] Rafaat H. Khalil & Nabil Al-Humadi, Types of Acute Phase Reactants and Their Importance in Vaccination (Review), 12 Biomed. Reps. 143 (2020).

[24] C-reactive protein is "a globulin that forms a precipitate with the somatic C-polysaccharide of the pneumococcus in vitro; it is the most predominant of the acute-phase proteins." C-Reactive Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100489 (last visited June 27, 2025).

[25] Transthyretin is "an α-globulin secreted by the liver that transports retinol-binding protein and thyroxine in the blood." Transthyretin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=50705 (last visited June 27, 2025).

[26] Cara L. Carty et al., Inflammatory Response After Influenza Vaccination in Men with and Without Carotid Artery Disease, 26 Arteriosclerosis Thrombosis & Vascular Biology 2738 (2006).

[27] Innate immunity is "immunity based on the genetic constitution of the individual." Innate Immunity, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=81992 (last visited June 27, 2025).

not identified in the list of reactants associated with vaccines in the paper from Khalil and Al-Humadi.

Dr. Monsell also cited a paper by Rogers et al.,[28] for the proposition that the NFKB gene was associated with an inflammatory process and expressed in both flu infections and with flu vaccinations. Pet. Ex. 63 at 7 (citing Pet. Ex. 91). NFKB was found to be one of 334 genes common to both flu infection and vaccination. Pet. Ex. 91 at 6. The authors noted that the NFKB signaling pathway "is activated during [flu] infection which up-regulates antiviral genes and can regulate viral synthesis." Id. at 11. The authors did not implicate NFKB as playing a role in causing hearing loss or SSNHL after the flu vaccine.

Of note, Rogers et al. also found that genes that were unique to flu infection and vaccination were "involved in different processes," which "indicate[d] that with the actual infection the body undergoes different processes to [those] induced by vaccination." Id. at 15-16.

II. Acute Phase Reactants reduce cochlear blood flow and cause vascular damage, including breakdown of the cochlear blood-tissue barrier.

Second, Dr. Monsell proposed that acute phase reactants reduced cochlear blood flow and caused vascular damage and breakdown of the cochlear blood-tissue barrier.[29] Pet. Ex. 63 at 6-7. He posited that "[f]ibrinogen is released in response to the [flu] vaccination and raises blood viscosity." Id. at 7. However, Khalil and Al-Humadi did not identify fibrinogen as an acute phase reactant associated with the flu vaccine. See Pet. Ex. 76 at 5 tbl.II. Fibrinogen was identified with *Staphylococcus* ("*S.*") *aureus* capsular polysaccharide vaccines, not flu vaccines. Id.

Dr. Monsell cited articles[30] to support the proposition that fibrinogen levels have been found to be elevated in hearing loss patients compared to controls as well as animal studies inducing hearing loss following injection of fibrinogen. Pet. Ex. 63 at 8 (citing Pet. Ex. 92 at 1

[28] Lavida R.K. Rogers et al., Microarray Gene Expression Dataset Re-Analysis Reveals Variability in Influenza Infection and Vaccination, 10 Frontiers Immunology 1 (2019). This was also cited as Resp. Ex. A.1.

[29] For more information on the cochlear blood-tissue barrier, see Pet. Ex. 85 (Lingling Neng et al., Endothelial Cell, Pericyte, and Perivascular Resident Macrophage-Type Melanocyte Interactions Regulate Cochlear Intrastrial Fluid-Blood Barrier Permeability, 14 J. Ass'n for Rsch. Otolaryngology 175 (2013)).

[30] Dr. Monsell also cited a 2018 article by Oya et al., but it does not appear that this article was filed. See Pet. Ex. 63 at 8.

("Fibrinogen plasma levels were significantly increased in [sudden hearing loss] patients.");[31] Pet. Ex. 103 at 1 (inducing acute hearing loss by injecting fibrinogen in an animal study)).[32] However, Dr. Monsell cited no foundational evidence to show that the flu vaccine at issue here contains fibrinogen or that fibrinogen is increased after administration of a flu vaccine.

Further, Khalil and Al-Humadi did not show that the flu vaccination was associated with an acute phase reaction relative to TNF. See Pet. Ex. 76 at 5 tbl.II. Dr. Monsell cited a paper by Sharaf et al.[33] for the premise that "TNF decreases cochlear blood flow." Pet. Ex. 63 at 8 (citing Pet. Ex. 94). The animal study reported by Sharaf et al. took baseline measurements of cochlear blood flow before and after "surgically exposed microvessels were superperfused with TNF (5.0 ng/ml) for [five] minutes" resulting in significantly reduced blood flow. Pet. Ex. 94 at 1-2. The authors noted that "inflammation and vascular disturbances have been observed [] in the pathogenesis of SSNHL." Id. at 4. The authors proposed that TNF-induced vascular constriction may play a role in this type of hearing loss. Id. at 4-5. But the authors do not discuss vaccination or TNF in the context of vaccination.

In addition to fibrinogen and TNF, Dr. Monsell also asserted that the flu vaccine activates NFKB. Pet. Ex. 63 at 8. He opined that hemagglutinin, which is in the flu vaccine, "strongly activates the binding and transcription of NFκB," which "induces production of [TNF], which in turn induces the production of NFκB, potentially creating a circular, accelerat[ed] pathway of cytokine secretion." Id. However, Dr. Monsell did not provide support for the premise that the flu vaccine[34] induces the binding and transcription of NFKB. And if even if he did, Dr. Monsell has not shown by foundational evidence that NFKB plays a role in hearing loss associated with the flu vaccine. Thus, the relevance of NFKB here is not clear.

The same is true of TNF. Dr. Monsell stated that TNF is "released in response to [flu] vaccination" and that "TNF activation" occurs as part of the immune response to the flu vaccine.

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[31] Claudia Rudack et al., Vascular Risk Factors in Sudden Hearing Loss, 95 Thrombosis & Haemostasis 454 (2006).

[32] Bernhard G. Weiss et al., Drug-Induced Defibrinogenation as New Treatment Approach of Acute Hearing Loss in an Animal Model for Inner Ear Vascular Impairment, 38 Otology & Neurotology 648 (2017).

[33] Kariem Sharaf et al., Tumor Necrosis Factor-Induced Decrease of Cochlear Blood Flow Can Be Reversed by Etanercept or JTE-013, 37 Otology & Neurotology e203 (2016).

[34] Dr. Monsell cited Pahl and Baweuerle for support; however, the authors reported the "flu *virus* hemagglutinin [] activated NF-κB DNA binding and transcriptional activation." Pet. Ex. 46 at 1 (emphasis added) (Heike L. Pahl & Patrick A. Baeuerle, Expression of Influenza Virus Hemagglutinin Activates Transcription Factor NF-κB, 69 J. Virology 1480 (1995)). The flu vaccine was not discussed.

Pet. Ex. 63 at 8. He cited a paper by Szyszko et al.,[35] published in 2006, reporting on the cytokine response to whole and split virus flu vaccines. Pet. Ex. 100 at 1-2. Four inflammatory cytokines were detected after vaccination, including TNF-α. Id. at 4. Higher levels of TNF-α were found in the whole virus vaccine, "whereas [the] split virus vaccine [was] dominated [by] the [cytokine] Il-1β response." Id. at 4, 7. The study does not appear to support a finding that the split virus vaccine[36]—"[t]he most commonly used formulation today"—significantly elevated TNF-α levels or that TNF activation occurred. Id. at 1.

The other articles cited by Dr. Monsell related to TNF do not show that the flu vaccination increases levels of TNF. Bertlich et al.,[37] for example, noted that impaired cochlear blood flow has been associated with SSNHL, and in their animal study, TNF decreased blood vessel diameter. Pet. Ex. 66 at 1; see also Pet. Ex. 68 at 5;[38] Pet. Ex. 85. But the authors did not discuss the flu vaccine or suggest that it increased or activated TNF so as to affect cochlear blood flow.

Regarding CRP, Dr. Monsell stated it "inhibits endothelial nitric oxide [] synthase expression, which in turn reduces [nitric oxide] bioavailability and increases the production of ROS." Pet. Ex. 63 at 9. He did not explain the relevance of this sentence but cited to several articles. Id. Carty et al. explained that the proteins CRP, serum amyloid A, and IL-6 are inflammatory markers that "are associated with an increased risk of cardiovascular disease." Pet. Ex. 70 at 1. These proteins are also described as "key components of the [APR] to injury or infection," and the APR is regulated by the NFKB pathway, "which is activated systemically by cytokines." Id. The authors studied APR in white men with severe carotid[39] artery disease (43) and in controls (61). Id. The study sought to determine "whether APR predicts or varies with vascular disease status." Id. Using the seasonal flu vaccine as "a standard immune stimulus," measurements were taken to determine carotid artery disease status and changes in plasma levels of the proteins known to be markers of inflammation, including CRP. Id. The study found that "APR may trigger [] systemic and local (at the vessel wall) inflammation" that "may contribute to cardiovascular disease." Id. at 6. However, the authors limited their discussion of the findings

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[35] E. Szyszko et al., Impact of Influenza Vaccine Formulation with a Detailed Analysis of the Cytokine Response, 64 Scandinavian J. Immunology 467 (2006).

[36] Dr. Monsell did not describe the vaccine at issue here. However, the record shows that Petitioner received the Fluarix vaccine, a split virus vaccine. See Pet. Ex. 1; Resp. Ex. A.15 at 17 (package insert).

[37] Mattis Bertlich et al., Cochlear Pericytes Are Capable of Reversible Decreasing Capillary Diameter In Vivo After Tumor Necrosis Factor Exposure, 38 Otology & Neurotology e545 (2017).

[38] Martin Canis & Mattis Bertlich, Cochlear Capillary Pericytes, 1122 Advances Experimental Med. & Biology 115 (2019).

[39] Carotid "pertain[s] to the principal artery of the neck." Carotid, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=8129 (last visited July 7, 2025).

to cardiovascular disease. Id. They did not discuss the cochlear blood system, which, based on Dr. Monsell's description, is unique and quite complex. See Pet. Ex. 63 at 1.

Pepys and Hirschfield[40] also discussed CRP in the context of cardiovascular disease and provided information about the protein as an "acute-phase reactant," discussing its "possible role in pathogenesis of disease." Pet. Ex. 87 at 1. The article is limited to cardiovascular disease and again does not discuss the role of CRP as it relates to cochlear blood flow.

Falsey et al.[41] studied how the flu A infection affected CRP levels in seven young adults, 15 elderly patients, and 36 hospitalized adult patients and found CRP levels elevated during acute flu illness in hospitalized patients. Pet. Ex. 75 at 1. The study further showed that symptoms of dyspnea (shortness of breath), wheezing, and fever, were associated with higher CRP levels. Id.

And Liuba et al.[42] studied levels of brachial artery endothelial function and levels of CRP and fibrinogen using the flu vaccine as an inflammatory stimulus in eight healthy men. Pet. Ex. 36 at 1. They found the "[flu] vaccination caused a slight elevation in CRP . . . and fibrinogen . . . at [two] days, which completely resolved at 14 days." Id. The authors noted that "[s]imilar to other vaccines, [the flu] vaccine causes a short-lived inflammatory response that resolves within 48-72 hours after vaccination. Slight elevation in CRP, fibrinogen, and proinflammatory cytokines occurs during the inflammatory phase." Id. at 7 (internal citation omitted). However, this study was limited to cardiovascular disease; these observations were not described in the context of the inner ear, cochlear blood flow, or hearing loss. Further, no injury was described in association with slight elevations in these proteins.

In summary, relative to steps one and two of his mechanism, Dr. Monsell has not shown that the flu vaccine causes a significant increase in fibrinogen, TNF, NFKB, or other reactants, or that these reactants cause adverse effects, specifically reduced blood flow to the inner ear, vascular damage to the blood vessels in the inner ear, or breakdown of the cochlear blood-tissue barrier.

III. Reduction of cochlear blood flow results in cochlear ischemia.

The third step of Dr. Monsell's mechanism is that reduction of cochlear blood flow results in cochlear ischemia. Pet. Ex. 63 at 9. He described how reduction of cochlear blood

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[40] Mark B. Pepys & Gideon M. Hirschfield, C-Reactive Protein: A Critical Update, 111 J. Clinical Investigation 1805 (2003).

[41] Ann R. Falsey et al., Response of C-Reactive Protein and Serum Amyloid A to Influenza A Infection in Older Adults, 183 J. Infectious Diseases 995 (2001).

[42] Petru Liuba et al., Residual Adverse Changes in Arterial Endothelial Function and LDL Oxidation After a Mild Systemic Inflammation Induced by Influenza Vaccination, 39 Annals Med. 392 (2007). This article was also cited as Resp. Ex. A.13.

flow causes vascular damage (including breakdown of the cochlear blood-tissue barrier) and cochlear ischemia. Id. at 6-9.

Dr. Monsell, however, has not shown that the flu vaccine reduces blood flow in the inner ear, causes vascular damage to the blood vessels in the inner ear, or leads to breakdown of the cochlear blood-tissue barrier. Therefore, there is no foundational evidence underlying this step of his proposed mechanism.

Further, the medical literature cited by Dr. Monsell does not show that the flu vaccine reduces cochlear blood flow or causes cochlear ischemia. For example, Brown et al.[43] conducted an animal study showing that transient asphyxia[44] caused by "interrupting respiration for brief periods" caused reversible changes in all measured cochlear potentials. Pet. Ex. 67 at 1, 3. The relevance of this study is not clear, as there is no evidence here that the flu vaccine induced interrupted respiration or caused oxygen deprivation. Nor is there any suggestion that presenting signs and symptoms included shortness of breath or respiratory compromise.

Dr. Monsell cited several other articles in support of this step. Upon review, these papers also do not support the premise that the flu vaccination reduces blood flow or otherwise causes or contributes to cochlear ischemia. Onal et al.[45] explained that the cochlea is "very sensitive to alterations in blood circulation, and transient ischemia of the cochlea may result in . . . noise-induced [hearing loss] and [SSNHL]." Pet. Ex. 86 at 1. The authors discussed the concepts of oxidative stress and ischemia and reperfusion injury and their role in hearing loss; however, they did not identify the flu vaccination as a trigger for transient ischemia of the cochlea.

Randolph et al.[46] described how repeated compression of local blood flow to animal cochlea induces "transient cochlear hypoxia." Pet. Ex. 90 at 1. Compression of the blood flow caused vascular spasm and a permanent decrease in blood flow. Id. at 2. Again, this study describes facts and circumstances not present in this case.

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[43] M. Christian Brown et al., Cochlear Inner Hair Cells: Effects of Transient Asphyxia on Intracellular Potentials, 9 Hearing Rsch. 131 (1983).

[44] Asphyxia refers to the "pathologic changes caused by lack of oxygen." Asphyxia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4494 (last visited July 7, 2025).

[45] Merih Onal et al., Ozone Prevents Cochlear Damages from Ischemia-Reperfusion Injury in Guinea Pigs, 41 Artificial Organs 744 (2007).

[46] H.-B. Randolf et al., Cochlear Blood Flor Following Temporary Occlusion of the Cerebellar Arteries, 247 Eur. Archives Otorhinolaryngology 226 (1990).

Nakashima et al.[47] provided an overview of disorders of cochlear blood flow, noting that while there have been reports of hearing loss thought to be caused by disturbances of blood flow in the cochlea, "direct evidence . . . is still lacking in most of the cases." Pet. Ex. 84 at 1. Of note, the authors explained that noise exposure has been associated with impaired cochlear blood flow. Id. at 4. The authors identified many different causes or contributors to hearing loss in addition to noise exposure, including endolymphatic hydrops,[48] changes that occur with aging, and nitric oxide, low blood flow, cochlear hemorrhage, tumor, blood flow disturbances caused by inner ear diseases such as Meniere's disease, and abnormalities of the posterior communicating arteries of the circle of Willis. Id. at 4-8. Specific to SSNHL, the authors noted that hearing loss has been associated with "increased fluid pressure in the inner ear," which is "considered to be associated with reduced cochlear blood flow." Id. at 7. Hearing loss has also been associated with "acute reduction of cerebrospinal fluid pressure," diseases that provoke infarcts (i.e., Susac syndrome, Takayasu's disease, polyarteritis, etc.), blood disorders (leukemia, sickle cell anemia, and polycythemia), and microembolism (reported after cardiopulmonary bypass surgery). Id. at 7-8. The authors did not report an association between vaccination and disturbances of cochlear blood flow leading to hearing loss.

IV. Cochlear ischemia results in an injury/reperfusion situation and generation of ROS, creating oxidative stress in cochlear hair cells.

While this statement may be supported by medical literature cited by Dr. Monsell, the articles cited by Dr. Monsell related to this step of his theory fail to show an association between vaccination and cochlear ischemia. And the literature cited by Dr. Monsell in support of this step does not bridge the causal gap between vaccination and cochlear ischemia.

For example, Mao and Chen[49] described the role of hair cells in the inner ear, and how impairment in function of hair cells leads to hearing loss secondary to noise exposure. Pet. Ex. 82 at 1. The authors described noise-induced hearing loss and its molecular pathogenesis, which may involve oxidative stress. Id. at 2-3. However, vaccination was not discussed by the authors.

[47] Tsutomu Nakashima et al., Disorders of Cochlear Blood Flow, 43 Brain Rsch. Program 17 (2003).

[48] "Endolymphatic hydrops is a condition in which too much endolymph is present." Pet. Ex. 84 at 5. Endolymphatic hydrops is also referred to as Meniere's disease, which is "hearing loss, tinnitus, and vertigo resulting from nonsuppurative disease of the labyrinth with edema." Meniere Disease, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70588 (last visited July 7, 2025).

[49] Huanyu Mao & Yan Chen, Noise-Induced Hearing Loss: Updates on Molecular Targets and Potential Interventions, 2021 Neural Plasticity 1.

Merchant et al.[50] discussed the pathology and pathophysiology of idiopathic SSNHL. Pet. Ex. 40. At the outset of their paper, they stated that the "cause and pathogenesis" of this type of hearing loss is unknown. Id. at 1. The authors described temporal bone histopathology from 17 subjects and found "the most common abnormality . . . [was] atrophy of hair cells and supporting cells of the organ of Corti,[51] with variable involvement of other structures." Id. at 8. The data did not support vascular occlusion or viral infection as a common etiology. Id. An alternative and novel hypothesis was proposed by the authors: an "abnormal activation of cellular stress pathway involving [NFKB] within the cochlea." Id. More specifically, they posited that activation of the NFKB pathway "can result in production of inflammatory cytokines and other stress-related proteins that can disrupt the homeostatic balance of a cell or tissue." Id. The authors issued a caveat, however, noting that while this idea was consistent with the histopathological findings, there was no "direct proof in support of [their] theory." Id. at 9.

Relative to NFKB, Dr. Monsell cited studies that he asserted "demonstrate the possibility that [flu] vaccination could activate the NFKB system in the inner ear under certain conditions, thus triggering a damaging local inflammatory response." Pet. Ex. 16 at 15. However, the studies cited by Dr. Monsell related to flu infections, not the flu vaccination.

For example, Pahl and Baeuerle showed that expression of "the [flu] virus hemagglutinin (HA)[] activates NF-κB DNA binding and transcriptional activation." Pet. Ex. 46 at 1. They explained that "NF-κB induces transcription of a variety of cytokines released during [flu] virus infection," and thus, activation of NFκB "represents one mechanism by which [flu] virus infection increases cytokine transcription." Id. at 4. The authors suggested several examples in which this information could be clinically relevant, but the examples involve flu viral infections, such as viral pneumonia. Id. The authors did not discuss vaccination.

These studies, Dr. Monsell asserted, "demonstrate the possibility that [flu] vaccination could activate the NFKB system in the inner ear under certain conditions, thus triggering a damaging local inflammatory response." Pet. Ex. 16 at 15. However, Dr. Monsell did not provide any literature showing the flu vaccination, not flu infection, can stimulate the NFKB pathway.

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[50] Saumil N. Merchant et al., Pathology and Pathophysiology of Idiopathic Sudden Sensorineural Hearing Loss, 26 Otology & Neurotology 151 (2005).

[51] The organ of Corti "rest[s] on the basilar membrane of the cochlear duct[] [and] contains the auditory hair cells, special sensory receptors for hearing, as well as several types of supporting cells. Organum Spirale, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=94965 (last visited July 7, 2025).

V. Cochlear hair cells and ROS-capture mechanisms struggle with oxidative stress. Some hair cells may survive if the degree of injury is mild, in others, the regulatory systems (including NFKB) sense severe cellular damages. To mitigate widespread tissue destruction, caspase pathways are activated.

For support, Dr. Monsell cited to the "injury/reperfusion model" by Yang et al.,[52] which "show[ed] an important link between ischemia and inflammation in the cochlea." Pet. Ex. 63 at 11 (citing Pet. Ex. 105). Yang et al. studied the effects of temporary occlusion of the carotid artery (ischemia) for 30 or 60 minutes on auditory brainstem responses in mice. Pet. Ex. 105 at 1. Significant hearing loss occurred after occlusions in both groups, although "the longer the ischemia lasted, the worse the resultant hearing impairment." Id. at 6. The authors concluded that autophagy[53] played a protective role in "cell survival in the cochlea," although "excessive [ischemia/reperfusion] damage overwhelms the beneficial" effects and can lead to cell death. Id. at 7. Again, the facts are not comparable to those presented here as there was no evidence the vaccine caused a temporary occlusion of the carotid artery.

Next, Dr. Monsell opined that activation of NFKB induces the production of cytokines and "a defensive cellular inflammatory response that may, if severe, include activation of the apoptotic cascade (programed cell death) and SSNHL," as described by Merchant et al. and Adams.[54] Pet. Ex. 63 at 11 (citing Pet. Exs. 18, 40). As previously described, Merchant et al. involved histopathology of temporal bones to better understand the cause of hearing loss. Pet. Ex. 40 at 1. Merchant et al. also stated that the effects of NFKB activation include apoptosis as well as "protection from apoptosis," although the "nature of the response depends on the molecular mediators that trigger NFκB activation and the context in which the activation occurs." Id. at 8. This hypothesis was described as the "stress response hypothesis." Id. Vaccination was not discussed.

[52] Haidi Yang et al., The Protective Effect of Autophagy on Ischemia/Reperfusion-Induced Hearing Loss: Implications for Sudden Hearing Loss, 28 NeuroReport 1157 (2017).

[53] Autophagy is "the segregation and digestion of part of the cell's own cytoplasmic material within lysosomes." Autophagy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=5011 (last visited July 7, 2025).

[54] Joe C. Adams, Clinical Implications of Inflammatory Cytokines in the Cochlea: A Technical Note, 23 Otology & Neurotology 316 (2002). In another article from Adams et al., systemic inflammatory stress was induced by injection of endotoxin and acoustic trauma was induced by intense noise exposure and resulted in NFKB activation. Pet. Ex. 19 at 1-2 (J.C. Adams et al., Selective Activation of Nuclear Factor Kappa B in the Cochlea by Sensory and Inflammatory Stress, 160 Neurosci. 530 (2009)). The authors stated, "[c]learly systemic inflammation[] do[es] not routinely cause unilateral hearing loss." Id. at 8. They suggested that such hearing loss would require a secondary stress, resulting in compounding effects. Id. Vaccination was not discussed.

Adams discussed the role of NFKB in cochlear cells and the possibility that NFKB could play a role in "[SSNHL] associated with otosclerosis, otitis media, and central nervous system inflammation[] . . . by disrupt[ing] [] the normal balance of inflammatory cytokines within the spiral ligament." Pet. Ex. 18 at 1. But they did not discuss this possible mechanism in the context of vaccination.

The last article cited by Dr. Monsell in support of this step of his proposed mechanism was by Cabrera et al.,[55] which described how the NFKB1 gene may influence prognosis in patients with Meniere's Disease. Pet. Ex. 25 at 1. The authors described two variants in the NFKB1 gene associated with rapid hearing loss progression in patients who have unilateral SNHL. Id. Immune-mediated inner ear disease and its pathogenesis was also discussed. Id. at 1-2. Dr. Monsell stated that the findings in Cabrera et al. illustrated how the genetic variants described could be affected by inflammatory challenges such as vaccination. Pet. Ex. 63 at 11. However, vaccination was not discussed by Cabrera et al. Moreover, an underlying genetic variant has not been identified as relevant here.

VI. Caspase pathways activate the mechanisms of apoptosis, resulting in programmed death of hair cells and irreversible SSNHL. If the damage is severe enough, cell death by necrosis may occur.

Dr. Monsell described apoptosis and necrosis as it relates to severe hearing loss. In Van De Water et al.,[56] the authors described caspases and their role in "programmed cell death" of inner ear sensory cells. Pet. Ex. 101 at 2. Caspases are defined as a "family of cysteine proteases[57] . . . present within the cells of normal healthy tissue in inactive (procaspase) forms." Id. at 3. Some members of caspases are involved in "programmed cell death (apoptosis) of cells," while others are involved in the "regulation and execution of apoptosis of affected cells," which occurs as part of normal development or due to "a cell's response to a high level of internal injury (e.g. membrane lipid peroxidation) that results from exposure to oxidative stress." Id. The authors described the pathway of cell death that arises out of an "insult that generates oxidative stress with the creation of [ROS] and other free radicals [] that damage the cell's organelles and internal membranes resulting in mitochondrial membrane damage and loss of membrane potential." Id. at 4. While programmed cell death is a normal part of development of the inner ear and normal hearing, caspases may also be involved in "maldevelopment . . . and a hearing deficit." Id. at 6. Caspases also play a role in cell death in cases of ototoxicity, as

[55] Sonia Cabrera et al., Intronic Variants in the *NFKB1* Gene May Influence Hearing Forecast in Patients with Unilateral Sensorineural Hearing Loss in Meniere's Disease, 9 PLoS One e112171 (2014).

[56] TR Van De Water et al., Caspases, the Enemy Within, Their Role in Oxidative Stress-Induced Apoptosis of Inner Ear Sensory Cells, 25 Otology & Neurotology 627 (2004).

[57] Cysteine protease (endopeptidase) is "any of a group of endopeptidases containing at the active site a cysteine residue involved in catalysis; the group includes papain and several cathepsins." Cysteine Endopeptidase, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=12267 (last visited July 7, 2025).

demonstrated by gentamicin[58]-induced injury to vestibular hair cells resulting in hearing loss. Id. at 6-7. The authors also described how acoustic trauma could cause hair cell loss with cell death and activation of caspase. Id. at 9. Van De Water et al. did not describe cell death caused by vaccination or suggest that vaccination-induced activation of a caspase pathway results in death of hair cells or hearing loss.

In support of this step, Dr. Monsell again cited Merchant et al., specifically Case 2, which was a 57-year-old male with pre-existing Meniere's disease in the left ear who had sudden hearing loss in the right ear after having a "head cold" for three days. Pet. Ex. 63 at 12 (citing Pet. Ex. 40 at 5-6). The patient was hospitalized for anticoagulant treatment and subsequently had sudden left leg pain with loss of pulses. Pet. Ex. 40 at 6. Surgery was performed to remove clots, but his condition deteriorated, and he died six days later due to coronary artery occlusion. Id. A postmortem examination of his temporal bones showed "marked swelling with edema and vacuole formation within the cytoplasm and blurring of cell boundaries." Pet. Ex. 63 at 12 (quoting Pet. Ex. 40 at 6). Although the patient's history suggested a vascular cause of hearing loss, the histopathology did not support the etiology of vascular occlusion. Pet. Ex. 40 at 6-7. Instead, it showed "unusual and severe swelling of hair cells" as well as "swollen and shrunken areas of the organ of Corti," which was interpreted "as evidence that these cells were under severe osmotic stress." Id. at 7. The authors opined these findings were consistent with a "stress-induced" hypothesis. Id. They stated that "if such stress were irreversible, these cells would undergo apoptosis or necrosis, resulting in atrophy of the organ of Corti, which is the abnormality observed in the majority of cases of sudden deafness that do not recover." Id. Again, vaccination was not discussed.

VII. Microthrombosis in the cochlear capillaries and other events may exacerbate the injury process or impair recovery of the inner ear, or the inner ear may recover enough from thrombosis to avoid fibrosis and neogenesis of bone in the cochlear duct.

In describing the last step of his mechanism, Dr. Monsell suggested a list of things that could worsen the "injury process or impair recovery" in the setting of inflammation: (1) upregulation of adhesion molecules on endothelial surfaces; (2) increased generation of oxygen species facilitated by deficient bioactivity of endothelial nitric oxide; (3) accelerated oxidative modification of low-density lipoprotein; (4) creation of micro-atherosclerotic emboli; and (5) activation of thrombosis in the microcirculation of the cochlea. Pet. Ex. 63 at 12. For support, Dr. Monsell cited four articles.

Liuba et al. used the flu vaccine to simulate mild systemic inflammation to study the effect on vascular endothelium. Pet. Ex. 36 at 1. They reported that abnormalities "possibly

[58] Gentamicin is "an aminoglycoside antibiotic complex" that "is effective against a wide range of aerobic gram-negative bacilli . . . and some gram-positive bacteria." Gentamicin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=20074 (last visited July 7, 2025). Gentamicin is known to be ototoxic and to cause hearing loss. Ototoxicity, Cleveland Clinic, https://my.clevelandclinic.org/health/diseases/24769-ototoxicity (last reviewed Feb. 22, 2023).

involving decreased [nitric oxide] and increased intravascular oxidative burden" could occur and that these events "might be important" in playing a role in arterial disease. Id. at 7. Additional studies were recommended to "verify this hypothesis." Id. The vascular endothelium and vascular system of the inner ear were not studied or discussed.

Albertini et al.[59] discussed the role of lipoprotein oxidation in the development of atherosclerosis. Pet. Ex. 64. The article does not discuss vaccination or the inner ear. Esmon[60] described the interplay between inflammation and thrombosis. Pet. Ex. 74. Esmon also does not discuss vaccination or the inner ear. Yang et al.[61] described research about "stem cell[] related regenerative therapies to prevent degeneration and damage of hair cells and spiral ganglion neuron." Pet. Ex. 106 at 6. While the article mentions ROS and the proinflammatory environment it induces through activation of caspase-1 and proinflammatory cytokines, the context is not vaccination. The focus of the paper appears to be about ways to "reduce . . . apoptosis of hair cells by down-regulating proinflammatory mediators" via the administration of cochlear spiral ganglion progenitor cell-derived exosomes ("CSGPC-exomes"). Id. at 6-8.

### 2. Additional Supporting Opinions and Medical Literature

After identifying his seven-part mechanism and citing medical literature, Dr. Monsell offered additional opinions explaining aspects of his proffered mechanism. He opined that there are multiple mechanisms implicated in SSNHL, and he acknowledged that hearing loss is "rare in association with vaccination." Pet. Ex. 63 at 13. He explained that each step of his mechanism is "subject to individual differences in genetic predisposition, microvascular anatomy, and risk factors." Id. Dr. Monsell added that comorbidities, including hypertension, cardiovascular disease, obesity, and other factors such as aging, can "increas[e] susceptibility to vascular injury" as well as prognosis. Id. This individuality extends to "[i]ndividual immune responses and toxicity to vaccination," which "can vary significantly, including responses to [the flu] vaccination." Id. This range in individual responses is thought to be due to genetic differences. Id. Whole-genome sequencing is identifying new prognostic factors for recovery from SSNHL as demonstrated in a 2022 study by Yang et al.[62] Id. at 13-14 (citing Pet. Ex. 107). Yang et al. identified key "hub genes" that are "independent prognostic factors of SSNHL." Pet. Ex. 107 at 4.

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[59] R. Albertini et al., Oxidation of Low-Density Lipoprotein in Atherosclerosis from Basic Biochemistry to Clinical Studies, 2 Current Molecular Med. 579 (2002).

[60] Charles T. Esmon, Reprint of Crosstalk Between Inflammation and Thrombosis, 61 Maturitas 122 (2008).

[61] Tao Yang et al., Exosomes Derived from Cochlear Spiral Ganglion Progenitor Cells Prevent Cochlea Damages from Ischemia-Reperfusion Injury Via Inhibiting the Inflammatory Process, 386 Cell & Tissue Rsch. 239 (2021).

[62] Anni Yang et al., Uncovering Novel Prognostic Factors of Sudden Sensorineural Hearing Loss by Whole-Genome Sequencing of Cell-Free DNA, 18 J. Int'l Advances Otology 459 (2022).

In discussing treatment, Dr. Monsell asserted "inflammation [] has a prominent role in the causation of SSNHL" due to clinical response to anti-inflammatory steroid treatment. Pet. Ex. 16 at 14. "[C]orticosteriods are potent down-regulators of inflammatory mediators" that "may act by reducing inflammatory aspects of cell trauma caused by cell death and other potential intra-cochlear inflammatory pathways activated by ischemic injury." Pet. Ex. 63 at 14.

Lastly, Dr. Monsell discussed epidemiology studies. Pet. Ex. 63 at 15-16. He noted that Baxter et al., a study of more than eight million trivalent flu vaccinations, did not find an association between flu vaccinations and SSNHL. Id. at 15 (citing Pet. Ex. 24 at 1). According to Dr. Monsell, if a study involved "a larger population and sufficient power," an association might be found. Id. at 15-16. He opined that "[w]ithout a massive, controlled clinical trial," it is "not [] possible" to determine the accurate incidence of SSNHL after flu vaccination. Id. at 16. Reliance on Baxter et al., however, to "reject the possibility of occasional rare cases . . . would not be rational." Id.

He also cited a case report of SSNHL two days after flu vaccination.[63] Pet. Ex. 63 at 16 (citing Pet. Ex. 34).[64] Kolarov et al. described the case of a 79-year-old woman who developed bilateral SSNHL and vertigo two days after flu vaccination. Pet. Ex. 34 at 1. Of note, the patient had a significant medical history that included insulin-dependent diabetes, atrial fibrillation, and past thalamic stroke, but no recent infection. Id. at 1-2. Kolarov et al. also described a 17-year-old girl with bilateral hearing loss, dizziness, and tinnitus 14 hours after receipt of an H1N1 vaccination. Id. at 2. The authors opined that an undetected infection or disease may have caused her hearing loss, or that it was speculatively related to her flu vaccination. Id.

### ii. Althen Prong Two

Dr. Monsell concluded that "more likely than not," Petitioner's flu vaccination had a "substantial causal role" in his development of right-sided SSNHL due to "a combination of complex factors, primarily idiosyncratic microvascular and inflammatory, which were triggered by his vaccination." Pet. Ex. 16 at 9, 17. He opined that but for this vaccination, Petitioner would not have developed SSNHL. Id. at 17.

In response to the opinions of Respondent's experts, Dr. Monsell explained that he proposed a "micro-vascular" inflammatory mechanism, not a "large-vessel mechanism" as suggested by Drs. Ying and Staats. Pet. Ex. 63 at 17. Dr. Monsell agreed that vascular

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[63] Dr. Monsell stated that the article by Alsanosi reported two cases of SSNHL after H1N1 vaccination. Pet. Ex. 63 at 16 (citing Pet. Ex. 65 (A.A. Alsanosi, Influenza A (H1N1): A Rare Cause of Deafness in Two Children, 126 J. Laryngology & Otology 1274 (2012))). However, Alsanosi described two reports in children after H1N1 infections, not vaccinations. Pet. Ex. 65 at 1. The first child had bronchopneumonia as a complication of her infection, and the second child had a high fever; both had bilateral hearing loss. Id. at 1-2.

[64] Claudia Kolarov et al., Bilateral Deafness Two Days Following Influenza Vaccination: A Case Report, 15 Hum. Vaccines & Immunotherapeutics 107 (2019).

obstruction caused by a "thrombo-embolic stroke" is not applicable because Petitioner did not have hypertension, stroke, or myocardial infarction and he did not have "profound deafness." Id. Dr. Monsell also opined acute vascular occlusion of the inner ear (or "[c]omplete vascular occlusion by a large-vessel mechanism"), a stroke, hypertension, myocardial infarction, and infections can be excluded as alternative causes. Pet. Ex. 16 at 11-13; Pet. Ex. 63 at 17-18.

Dr. Monsell acknowledged Petitioner did not respond to oral steroids (prednisone), an accepted treatment for SSNHL. Pet. Ex. 16 at 15. However, he maintained that an "initial absence of response . . . does not indicate that inflammation was not part of the mechanism of SSNHL." Id.; see also Pet. Ex. 63 at 15 ("[A] lack of response to corticosteroid treatment in individual cases does not imply that their mechanisms of injury did not include inflammatory processes, just that the strength of the injury was too great for recovery."). Dr. Monsell hypothesized that treatment could fail for many reasons, and here, Petitioner's "SSNHL was in a guarded prognostic category from the beginning as indicated by the moderate to severe level of pure tone test results and his unmeasurable word recognition score." Pet. Ex. 16 at 15. Following Petitioner's transtympanic steroid injection, while still taking oral steroids, Petitioner's right ear showed improvement in pure tone hearing levels according to Dr. Monsell, despite not resulting in functional hearing improvement. Id.

Regarding Dr. Staats' argument that Petitioner's lack of MRI enhancement was evidence against an inflammatory cause of his SSNHL, Dr. Monsell attributed this to the fact that Petitioner's MRI study was not done using gadolinium contrast. Pet. Ex. 63 at 18-19 (citing Resp. Ex. C at 4-5). Dr. Monsell cited a study from Compagnone et al.[65] that described enhancement in the cochlea after contrast injection with gadolinium. Id. at 18 (citing Pet. Ex. 71 at 3). Dr. Monsell opined Petitioner's MRI was not performed using gadolinium, and therefore it was inappropriate for Dr. Staats to disregard an inflammatory and/or microvascular mechanism. Id. at 18-19. However, the medical records show that Petitioner's MRI "[was] performed both prior to and following the intravenous administration of . . . Dotarem," a "gadolinium-based contrast agent." Pet. Ex. 4 at 16; Dotarem, PDR, https://www.pdr.net/drug-summary/?drugLabelId=3146#description (last visited July 9, 2025). Therefore, Dr. Monsell is incorrect in stating Petitioner's MRI was not performed using gadolinium. And he is incorrect in asserting the enhancement seen in Compagnone et al. could not have been seen here.

Dr. Monsell acknowledged, however, that Petitioner's MRI was unremarkable and did not identify a cause of Petitioner's hearing loss. Pet. Ex. 63 at 18-19. He further defended his opinion that the MRI was not determinative since "[t]here is nothing in the [MRI] report to indicate that the radiologists were looking for the type of specialized information regarding SSNHL described by Compagnone [et al.]" Id. at 18-19.

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[65] L. Compagnone et al., Injected 3T-3D-FLAIR-MRI Labyrinthine Patterns Match with the Severity and Tonotopic Alteration in Sudden Sensorineural Hearing Loss, 279 Eur. Archives Oto-Rhino-Laryngology 4883 (2022). Compagnone et al. used an MRI protocol that included administration of gadolinium contrast, with specific optimized 3D-FLAIR sequences and parameters, including 10 minute and four hour delayed sequences. Pet. Ex. 71 at 2-3, 3 tbl.1. The study demonstrated the ability of MRI using the specified sequences and parameters to "reveal labyrinthine involvement in sudden hearing loss." Id. at 8.

Dr. Monsell acknowledged Petitioner received numerous prior flu vaccinations but asserted that "prior [flu] vaccinations should not be used as a point to argue in favor or against the proposed mechanism." Pet. Ex. 63 at 15. He explained every vaccine is different and the human body is always changing, thus "susceptibility to SSNHL is not fixed." Id.

Dr. Monsell opined that alternative factors were not the cause of Petitioner's SSNHL. Pet. Ex. 16 at 8-13. First, he explained Petitioner did not have eustachian tube dysfunction. Id. at 9. Although Petitioner's first visit following onset resulted in an assessment of obstruction of the right eustachian tube on October 26, 2018, Dr. Monsell noted this diagnosis was excluded by Petitioner's treating physicians once Petitioner received audiometric testing and saw otolaryngologist Dr. Holm. Id. (citing Pet. Ex. 2 at 105, 107, 115).

Dr. Monsell next ruled out age-related[66] and noise-induced hearing loss as both typically occur gradually and cause bilateral hearing loss, while Petitioner's SSNHL was sudden and unilateral. Pet. Ex. 16 at 10. Petitioner was not on ototoxic medication, nor did Petitioner have Meniere's disease, both of which can result in hearing loss. Id. at 11.

While Dr. Monsell agreed Petitioner did have mild dyslipidemia, for which he was taking medication (Zocor), and noted dyslipidemia has been associated with SSNHL in some studies, he asserted Petitioner had no other clinical signs or symptoms consistent with vascular disease that would predispose Petitioner to a stroke or similar event. Pet. Ex. 16 at 11.

Next, Dr. Monsell agreed Petitioner did not have autoimmune inner ear disease ("AIED"). Pet. Ex. 16 at 10-11. He explained AIED occurs when "the body's immune system makes a mistake, attacking a normal organ or molecule in the body as though it were a foreign invader." Id. at 10. Petitioner's presentation (sudden, unilateral, and profound from onset) and lack of response to steroid treatment, according to Dr. Monsell, does not correspond to clinical findings in AIED, and thus, this diagnosis can be excluded. Id. Nevertheless, Dr. Monsell asserted "AIED is not unrelated to [Petitioner's] case" since AIED "establishes that a disease process that is primarily inflammatory—and not infectious—can cause severe SNHL." Id. at 10-11.

Lastly, Dr. Monsell disagreed that Petitioner had an asymptomatic viral infection that caused his hearing loss. Pet. Ex. 16 at 12. Petitioner did not have signs or symptoms of infection associated with hearing loss. Id. Further, his MRI was normal and did not show enhancement of the applicable cranial nerve which occurs with viral-related inflammation of the inner ear. Id. at 13.

[66] Dr. Monsell opined it was "possible" that Petitioner had age-related hearing loss prior to vaccination. Pet. Ex. 16 at 10. He added, "[a]lthough age-related hearing loss was not the primary cause of [Petitioner's] SSNHL, it may have been a disease factor that made his right ear more susceptible to vaccine related SSNHL." Id.

#### iii. Althen Prong Three

Petitioner received a flu vaccination on October 23, 2018, and two days later, on October 25, he experienced sudden hearing loss in his right ear, later diagnosed as SSNHL. Pet. Ex. 16 at 17; Pet. Ex. 63 at 16. Dr. Monsell opined this two-day onset post-vaccination is consistent with his proposed mechanism as it is "well within the period of maximal activity of the [APR] to his vaccination." Pet. Ex. 63 at 12, 15-16 ("Sudden onset suggests some form of vascular mechanism as with the proposed mechanism . . . ."); see also Pet. Ex. 16 at 17.

He further noted that the 1994 Institute of Medicine ("IOM") report[67] accepted the "timing of an immune-mediated response following vaccination up to 42 days after vaccination." Pet. Ex. 16 at 17 (citing Pet. Ex. 54 at 3); see also Pet. Ex. 63 at 16. For further support, he cited Baxter et al., who "reported [three] cases of sudden hearing loss within [one] week among subjects who received vaccination for H1N1 [flu]." Pet. Ex. 16 at 17 (citing Pet. Ex. 24).

While Dr. Monsell acknowledged temporal association alone is insufficient to establish a causal connection, he stressed it "cannot be dismissed as definitively coincidental." Pet. Ex. 63 at 16. Dr. Monsell concluded that his theory "is consistent with the medically accepted timeframe for vaccine-related adverse events." Pet. Ex. 16 at 17; Pet. Ex. 63 at 16.

### 2. Respondent's Expert, Dr. Herman F. Staats[68]

#### a. Background and Qualifications

Dr. Staats obtained his B.S. in medical technology from Salisbury University in Salisbury, Maryland and a Ph.D. in basic medical sciences (microbiology and immunology) from the University of South Alabama in Mobile, Alabama. Resp. Ex. B at 1. He is a professor in the Department of Pathology at Duke University School of Medicine and holds joint appointments as an associate professor of immunology and associate professor of medicine. Id.; Resp. Ex. A at 1. He is also a member of the Duke Human Vaccine Institute. Resp. Ex. A at 1. Throughout his career, Dr. Staats has been a member of and/or held positions with various organizations and professional societies. Resp. Ex. B at 3-4. Dr. Staats "perform[s] research on the discovery and evaluation of vaccine adjuvants." Resp. Ex. A at 1. His bibliography consists of over 200 publications. Resp. Ex. B at 22-35.

#### b. Opinion

Dr. Staats opined that the "scientific literature does not support the mechanism of vaccine-induced hearing loss proposed by Dr. Monsell." Resp. Ex. A at 1.

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[67] Inst. of Med., Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality (Kathleen Stratton et al. eds., 1994).

[68] Dr. Staats provided two expert reports. Resp. Exs. A, E.

### i. Althen Prong One

First, Dr. Staats discussed "[a]ctivation of the innate immune system (inflammation) by vaccination." Resp. Ex. A at 2. Although Dr. Staats agreed that vaccines induce an inflammatory process, he explained "it is important to distinguish between inflammation induced by [flu] vaccination, inflammation included by [flu] infection, and inflammation induced by [lipopolysaccharide endotoxin ("LPS")[69]], both in terms of the magnitude of inflammatory response, and in terms of biological processes . . . related to inflammation-induced hearing loss." Id.

For example, Dr. Monsell opined that the NFKB pathway regulates inflammation in blood vessels in the ear. Resp. Ex. A at 2 (citing Pet. Ex. 16 at 15). However, the literature cited by Dr. Monsell discussed activation of the NFKB pathway after infection with viral or bacterial pathogens, not vaccination. Id. (citing Pet. Ex. 16 at 15); see, e.g., Pet. Ex. 45 at 1 ("In many cell types, nuclear NF-κB activity is induced by exposure to a wide variety of bacteria or bacterial products" and "[m]any viruses [] induce NFκB activity.").[70]

In his second report, Dr. Staats showed that flu vaccination and flu infection do not lead to the same inflammatory profile. Resp. Ex. E at 5-6. Masuda et al.[71] investigated inflammatory biomarkers in SSNHL and found that IL-6 was elevated. Resp. Ex. E.4 at 1. Bian et al.[72] reported elevated levels of cytokine IL-6 after acute flu A infection. Resp. Ex. E.6 at 1. However, Talaat et al.[73] showed no change in IL-6 levels following flu vaccination.[74] Resp. Ex. E.7 at 4. But see Resp. Ex. A.3 at 3 (reporting an increase of IL-6 by 0.70 pg/mL at 24 hours

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[69] LPS is "a Gram-negative bacterial component that is widely used to study inflammatory processes" in animal studies. Pet. Ex. 18 at 2.

[70] Heike L. Pahl, Activators and Target Genes of Rel/NF-κB Transcription Factors, 19 Oncogene 6853 (1999). The authors also provided a table of "[i]nducers of NF-κB activity." Pet. Ex. 45 at 2 tbl.1.

[71] Masatsugu Masuda et al., Correlations of Inflammatory Biomarkers with the Onset and Prognosis of Idiopathic Sudden Sensorineural Hearing Loss, 33 Otology & Neurotology 1142 (2012).

[72] Jia-Rong Bian et al., Clinical Aspects and Cytokine Response in Adults with Seasonal Influenza Infection, 7 Int'l J. Clinical & Experimental Med. 5593 (2014).

[73] Kawsar R. Talaat et al., Rapid Changes in Serum Cytokines and Chemokines in Response to Inactivated Influenza Vaccination, 12 Influenza & Other Respiratory Viruses 202 (2018).

[74] The authors of the study noted that subject eight had a "distinctively robust change" in cytokines, including IL-6. Resp. Ex. E.7 at 5.

after vaccination although 30% had a delayed response or no increase in IL-6 post-vaccination).[75]

Further, Dr. Staats cited a study by Rogers et al., which found there were "differentially expressed genes" after flu infection as compared to vaccination. Resp. Ex. A at 2 (citing Pet. Ex. 91 at 1). Dr. Staats quoted the following from Rogers et al.:

> Following pathway enrichment, we observed that the genes that are unique to each disease state ([flu] infected and vaccinated) are involved in different processes. For example, the biologically significant genes only in [flu] infected samples were enriched in pathways such as neutrophil degranulation and cell cycle checkpoints (SDF21 of online supplementary data files). Neutrophil degranulation is a defensive process neutrophils undergo to protect the host against invading pathogens. On the other hand, pathways involved in interferon signaling and antigen processing were enriched for the genes only in the vaccinated gene list (SDF22 of online supplementary data files). This indicates that with the actual infection the body undergoes different processes to that induced by vaccination.

Id. at 2-3 (quoting Pet. Ex. 91 at 15-16).

Rogers et al. noted that "one of the top 10 up-regulated genes expressed following [flu] infection" was OTOF, a gene that encodes the protein otoferlin. Resp. Ex. A at 3 (citing Pet. Ex. 91 at 11 tbl.2). Mutations in this gene have been associated with hearing loss. Id. Dr. Staats pointed out that "neither the expression of OTOF, nor the expression of other genes involved in hearing, are among the top 10 up-regulated genes identified following [flu] immunization." Id. (citing Pet. Ex. 91 at 11 tbl.2).

Regarding inflammatory biomarkers,[76] a recent (2023) literature review by Al-Azzawi and Stapleton,[77] cited by Dr. Staats, found that "[f]ibrinogen levels were greater in the non-recovery group compared with the control group and were higher in successive bilateral hearing loss than [in] unilateral [SSNHL]." Resp. Ex. E.1 at 3, 5. The authors also reported elevated "inflammatory markers, suggesting an almost universal inflammatory process." Id. at 4. However, CRP levels were non-specific and added little information about etiology, "beyond identification of inflammation." Id. The authors were unable to identify the causes or risk factors for SSNHL, recommending future focused investigations "on a case-by-case basis" to uncover "metabolic, infective[,] and autoimmune conditions." Id. at 7.

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[75] Arielle S. Radin et al., Using the Influenza Vaccine as a Mild, Exogenous Inflammatory Challenge: When Does Inflammation Peak?, 13 Brain Behavior & Immunity Health 1 (2021).
[76] For the significance of inflammatory biomarkers, see Resp. Ex. E.4.

[77] A. Al-Azzawi & E. Stapleton, Blood Tests as Biomarkers for the Diagnosis and Prognosis of Sudden Sensorineural Hearing Loss in Adults: A Systematic Review, 137 J. Laryngology & Otology 977 (2023).

Next, Dr. Staats distinguished the study cited by Dr. Monsell in support of his position that the flu vaccine can induce an inflammatory response in the inner ear leading to hearing loss. Resp. Ex. A at 4 (citing Pet. Ex. 16 at 14). Dr. Staats explained that Thomas and Moridani[78] does not discuss inflammatory reactions induced by vaccines, or discuss the inner ear, or hearing loss. Id. (citing Pet. Ex. 56). Further, studies cited by Dr. Monsell in support of inflammation-induced hearing loss use methodology which is not comparable to vaccination. Id. at 5. For example, in the animal study conducted by Adams et al., LPS from *Escherichia* ("*E.*") *coli* was injected in doses sufficient to model septic shock. Id. (citing Pet. Ex. 19 at 3). As noted by Radin et al., "vaccines lead to increases in circulating cytokines in the range of 1 pg/ml," while "endotoxin administration[] [] results in 100-fold increases in circulating pro-inflammatory cytokines." Id. at 6 (quoting Resp. Ex. A.3 at 1). Dr. Staats concluded that the "literature cited by Dr. Monsell suggesting that systemic inflammation can trigger the release of cytokines sufficient to cause hearing loss does not provide a meaningful analogy [of] the barely detectable cytokine concentrations observed following repeated [] [flu] vaccinations." Id.

Moving to Dr. Monsell's theories based on vascular changes secondary to vaccination, Dr. Staats explained why Liuba et al. is not informative here. Resp. Ex. A at 6 (citing Pet. Ex. 36). Notably, the flu vaccine in Liuba et al., Vaxigrip, has been administered for almost "50 years in over 120 countries with more than 1.8 million doses distributed" and hearing loss has not been identified as an adverse reaction. Id. (citing Pet. Ex. 36; Resp. Ex. A.14 at 2, 13-14).[79] Hearing loss has also not been identified as an adverse event associated with the flu vaccine at issue here, Fluarix. Id. (citing Resp. Ex. A.15 at 2, 14-15 (package insert)).

Dr. Staats also cited studies which have failed to find an association between the flu vaccine and vascular events and/or cardiac function. Resp. Ex. A at 7 (citing Resp. Ex. A.16 at 1 ("[N]o increase in the risk of myocardial infarction or stroke in the period after [flu], tetanus, or pneumococcal vaccination" although an increased risk for these was found "after a diagnosis of systemic respiratory tract infection.");[80] Resp. Ex. A.17 at 1 ("[V]ascular and cardiac functions were largely unaltered" after stimulation of "systemic inflammation via [flu] vaccination.").[81]

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[78] Chandan Thomas & Majid Moridani, Interindividual Variations in the Efficacy and Toxicity of Vaccines, 278 Toxicology 204 (2010).

[79] Margaret Haugh et al., A Trivalent, Inactivated Influenza Vaccine (Vaxigrip®): Summary of Almost 50 Years of Experience and More Than 1.8 Billion Doses Distributed in Over 120 Countries, 16 Expert Rev. Vaccines 545 (2017).

[80] Liam Smeeth et al., Risk of Myocardial Infarction and Stroke After Acute Infection or Vaccination, 351 New Eng. J. Med. 2611 (2004).

[81] Alexandra M. Coates et al., Exercise Alters Cardiac Function Independent of Acute Systemic Inflammation in Health Men, 320 Am. J. Physiology Heart & Circulatory Physiology H1762 (2021).

Regarding the colloquy between Dr. Monsell and Dr. Staats about micro-vascular versus macro-vascular (large blood vessel disease), Dr. Staats explained that he cited the paper by Smeeth et al. to illustrate the differences between infection and vaccination. Resp. Ex. E at 3 (citing Resp. Ex. A.16). Dr. Staats reiterated that Smeeth et al. concluded "acute infections are associated with a transient increase in the risk of vascular events." Id. (quoting Resp. Ex. A.16 at 1). "By contrast," Smeeth et al. added, "[flu] . . . vaccinations do not produce a detectable increase in the risk of vascular events." Id. (quoting Resp. Ex. A.16 at 1). Dr. Staats acknowledged that Smeeth et al. did not address micro-vascular inflammatory disease or its association with the flu vaccination or SSNHL. Id. at 3-4 (citing Resp. Ex. A.16).

Dr. Staats concluded by noting that the literature does not support "[flu] vaccine-induced micro-vascular inflammation as a cause of SSNHL." Resp. Ex. E at 4.

### ii. Althen Prong Two

Dr. Staats opined that "there is no evidence in [Petitioner's] medical records to support the conclusion that his hearing loss was due to the [flu] vaccine."[82] Rep. Ex. E at 1.

First, Dr. Staats discussed an alternative cause for Petitioner's hearing loss: asymptomatic infection. Resp. Ex. A at 4-5. Dr. Staats observed that Dr. Monsell excluded viral infections as the cause of Petitioner's hearing loss. Id. (citing Pet. Ex. 16 at 12-13). However, Dr. Monsell also cited to a case report of an asymptomatic patient who suffered profound hearing loss due to a subclinical mumps infection. Id. at 4 (citing Pet. Ex. 16 at 12; Pet. Ex. 44).[83] As illustrated by Otake et al., a patient can be infected with a virus, have no clinical symptoms, and still develop hearing loss. Pet. Ex. 44 at 1. Infections other than mumps associated with hearing loss include enterovirus infection. Resp. Ex. E at 2-3 (citing Resp. Ex. E.3 at 2 ("Twenty two (40%) of the 55 serum samples obtained from patients with sudden hearing loss contained enterovirus RNA . . . .")).[84] Thus, Dr. Staats explained that a viral etiology cannot be excluded, even though Petitioner did not report signs and symptoms of a viral infection. Resp. Ex. A at 4-5; Resp. Ex. E at 3.

Second, Dr. Staats opined that Petitioner's high cholesterol was a risk factor. Resp. Ex. A at 9. He cited studies showing that patients with idiopathic hearing loss had elevated

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[82] In addition to the opinions summarized here, Dr. Staats also discussed "evaluated blood inflammatory parameters, including [] [] neutrophils and lymphocytes" found in patients with SSNHL. Resp. Ex. A at 8. He noted that Petitioner's medical records did not include a complete blood count ("CBC") which would have measured these markers. Id. As these markers were not measured, the undersigned finds this opinion speculative.

[83] Hironao Otake et al., 3D-FLAIR Magnetic Resonance Imaging in the Evaluation of Mumps Deafness, 70 Int'l J. Pediatric Otorhinolaryngology 2115 (2006).

[84] Renate Mentel et al., Are Enterovirus Infections a Co-Factor in Sudden Hearing Loss?, 72 J. Med. Virology 625 (2004).

triglycerides. Id. at 8 (citing Resp. Ex. A.21 at 1 (concluding "[h]ypertriglyceridemia and low levels of [high-density lipoprotein-cholesterol] may be important factors in the pathogenesis of SSNHL");[85] Resp. Ex. A.22 at 2-4 (finding "auditory dysfunction [] associated with an increased in lipid profile atherogenicity")).[86] In Kaneva et al., elevated levels of the atherogenic ("ATH") index[87] were seen in patients with idiopathic SSNHL, although "most patients with idiopathic SSNHL did not have hyperlipidaemia." Resp. Ex. A.22 at 4-5. The authors found that "subjects with unfavourable ATH index values had a more than [four] times higher risk of idiopathic SSNHL" and they concluded that "high values of the ATH index were significantly associated with an increased risk of SSNHL." Id. at 4, 6. Based on Petitioner's chronic high cholesterol and age (62),[88] Dr. Staats opined that "it seems possible that [Petitioner's] hearing loss is associated with his chronic condition of hypertriglyceridemia." Resp. Ex. A at 8. In his second report, Dr. Staats cited to the recent (2023) literature review by Al-Azzawi and Stapleton in further support of this opinion. Resp. Ex. E. at 1 (citing Resp. Ex. E.1 at 3 ("Low-density lipoprotein (LDL) levels were . . . higher in [SSNHL] . . . .").

His opinions in this regard were informed by the medical literature, as well as references in Petitioner's medical records noting elevated triglycerides. See Resp. Ex. A at 9 (citing Pet. Ex. 2 at 14, 56, 113, 187 (records documenting or discussing Petitioner's high cholesterol)).

The third opinion by Dr. Staats relevant to Althen prong two was based on Petitioner's MRI, which was normal and did not show inflammation in his inner ear. Resp. Ex. A at 5, 9. Dr. Staats opined that if Dr. Monsell's theory of inflammation was accurate, the absence of inflammation suggests this was not a cause of his hearing loss. Id.

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[85] Mir Mohammad Jalali & Mahsa Nasimidoust Azgomi, Metabolic Syndrome Components and Sudden Sensorineural Hearing Loss: A Case-Control Study, 277 Eur. Archives Oto-Rhino-Laryngology 1023 (2020).

[86] Anastasiya Kaneva et al., The Atherogenic Index (ATH Index) As a Potential Predictive Marker of Idiopathic Sudden Sensorineural Hearing Loss: A Case Control Study, 18 Lipids Health & Disease 1 (2019).

[87] "The ATH index is a compound index of lipid metabolism . . . calculated by using values of both lipids and apolipoproteins." Resp. Ex. A.22 at 6. Apolipoproteins are "protein constituents of lipoproteins." Apolipoprotein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=3779 (last visited July 7, 2025). The ATH index was calculated using a formal including measures of plasma total cholesterol ("TC"), high density lipoprotein cholesterol ("HDL-C"), and apolipoprotein B and apolipoprotein A-I containing lipoproteins ("apo"). Resp. Ex. A.22 at 3.

[88] Dr. Staats noted that SSNHL is more commonly seen in those in their 60s and 70s. Resp. Ex. A at 8 (citing Resp. Ex. A.19 at 1 (Mehmet Eser Sancaktar et al., The Prognostic Value of Circulating Inflammatory Cell Counts in Sudden Sensorineural Hearing Loss and the Effect of Cardiovascular Risk Factors, 99 Ear Nose & Throat J. 464 (2020)).

#### iii. Althen Prong Three

Dr. Staats did not dispute that there was a temporal association between Petitioner's hearing loss and the administration of his flu vaccination, but he opined that the posited mechanism was "not supported by the published literature." Resp. Ex. A at 9.

### 3. Respondent's Expert, Dr. Yu-Lan Mary Ying[89]

#### a. Background and Qualifications

Dr. Ying is "a fellowship trained otologist/neurotologist[] and [is] board certified in otolaryngology and neurotology." Resp. Ex. C at 1. After she obtained in M.D. in New York from State University of New York ("SUNY"), Stony Brook School of Medicine, she completed a general surgery internship and otolaryngology residency at the University of Pittsburgh Medical Center. Resp. Ex. D at 1. Dr. Ying also completed a fellowship at the Howard Hughes Medical Institute-National Institute of Health during medical school as well as a post-graduate fellowship in otology/neurotology at Pittsburgh Ear Associates and a post-graduate fellowship in neurotology at Baylor College of Medicine. Id. at 1-2. Since 2014, Dr. Ying has worked as an assistant professor in the Otolaryngology-Head and Neck Surgery Department at Rutgers-New Jersey Medical School. Id. at 2. She has also held various hospital appointments and memberships in various professional societies, and she has served on several committees. Id. at 2-5. Dr. Ying has "a busy academic clinical practice treating many diverse patients with hearing, balance[,] and lateral skull base disorders." Resp. Ex. C at 1. She has co-authored over 30 publications throughout her career. Resp. Ex. D at 7-9.

#### b. Opinion

Dr. Ying explained that SSNHL is defined as a "hearing loss of 30dB or greater over at least three contiguous audiometric frequencies occurring within a 72-hr period." Resp. Ex. C at 4. Most people with SSNHL have "no identifiable cause" and are therefore classified as "idiopathic." Id. The most frequent causes are infection, otologic, traumatic, vascular, hematologic, and neoplastic. Id. Infectious causes include viruses, such as herpes simplex, varicella zoster, enteroviruses, and flu. Id. However, the most common viral cause is mumps, which causes endolymphatic labyrinthitis. Id. For patients with an unknown cause (idiopathic), many different etiologies have been suggested. Id. Dr. Ying stated that the "most widely accepted theories" include "vascular compromise, cochlear membrane rupture, and viral infection." Id. (internal citations omitted).

Based on her review of the records, Dr. Ying opined there is "insufficient evidence" to show Petitioner's SSNHL was "due to an adverse reaction to [the] flu vaccination." Resp. Ex. C at 4.

[89] Dr. Ying provided two expert reports. Resp. Exs. C, F.

### i. Althen Prong One

Dr. Ying opined "there is no reliable scientific evidence indicating [SSNHL] is an adverse event to vaccination," especially to an inactivated flu vaccination. Resp. Ex. C at 7; see also Resp. Ex. F at 4-5.

Fundamentally, Dr. Ying took issue with Dr. Monsell's assertion that inflammation in the inner ear was induced by the NFKB system and activated by "viral infection or immunization." Resp. Ex. C at 5 (quoting Pet. Ex. 16 at 15). There are several reasons she disagreed with this proposition.

First, she disagreed that the literature cited by Dr. Monsell supported his proposed mechanism. Resp. Ex. C at 5. The literature described "experimental conditions" based on animal studies that were very different than the conditions present here (inactivated flu vaccination in an adult human). Id. (citing Pet. Exs. 18, 40). Adams used intraperitoneal injection of LPS in mice to activate the NFKB system, conditions not comparable to vaccination. Id. (citing Pet. Ex. 18). The flu vaccine cannot be compared to LPS endotoxin, a "gram negative bacterial component" used in Adams. Id.; Resp. Ex. F at 4 (citing Pet. Ex. 18). Dr. Ying emphasized that "LPS-induced inflammation is not a comparable model that is applicable to vaccine-induced inflammation." Resp. Ex. F at 4.

Next, regarding Merchant et al., Dr. Ying explained the authors "did not identify vaccination as a possible trigger for activation of NF-κB." Resp. Ex. C at 5 (emphasis omitted). None of the cases studied "had a history of vaccination prior to sudden onset of hearing loss." Id. Both studies concluded more information was needed to understand the potential role of NFKB and related cytokines within cochlear cell types and their significance to cochlear pathologies. Id. (citing Pet. Exs. 18, 40). And Merchant et al. noted that their hypothesis lacked "any direct proof in support [of the] theory." Id. (quoting Pet. Ex. 40 at 10). In conclusion, Dr. Ying stated that "[t]o date, there have been no further studies to analyze activation of NFκB within the cochlea." Id.

Third, Dr. Monsell's theory based on NFKB relied on studies that involve flu infections, not vaccinations. Resp. Ex. C at 5. Dr. Ying opined "Dr. Monsell has not, and cannot, explain why a live infection is comparable to flu vaccination." Id. She explained that "[flu] vaccines cannot cause flu illness because they contain 'inactivated' (killed) virus, attenuated (weakened) virus, or are made using recombinant methods that do not use flu virus in the manufacturing process." Id. Additionally, the flu vaccine does not contain the bacterial LPS endotoxin used in Adams et al., and "a similar outcome should not be assumed." Id. (citing Pet. Ex. 18).

Dr. Monsell opined that the studies he cited, "demonstrate the possibility that [the flu] vaccination could activate the NFKB system in the inner ear under certain conditions thus triggering a damaging local inflammatory response." Resp. Ex. C at 6 (quoting Pet. Ex. 16 at 15). Dr. Ying observed that Dr. Monsell did not define what he meant by the phrase "certain conditions." Id.

Fourth, she disagreed with Dr. Monsell's conclusion that Baxter et al. was "underpowered to detect association or causation in rare instances." Resp. Ex. C at 6-7 (quoting Pet. Ex. 16 at 16). Dr. Ying noted that the authors specified that they were "able to provide strong evidence against an association" between the flu vaccine and hearing loss even with "over [eight] million doses." Id. (quoting Pet. Ex. 24 at 5).

The fifth reason is based on Dr. Monsell's references to literature purporting to support the premise that fibrinogen is released in response to flu vaccination. Resp. Ex. F at 1-2. Dr. Monsell stated, "[f]ibrinogen is released in response to [flu] vaccination." Id. (quoting Pet. Ex. 63 at 7). He cited a study by Rudack et al. and stated that the authors reported "serum fibrinogen levels were significantly higher in SSNHL patients compared to age- and sex- matched controls." Pet. Ex. 63 at 8 (citing Pet. Ex. 92 at 5). However, as noted by Dr. Ying, Rudack et al. stated that while "the mean fibrinogen concentration . . . was significantly increased in [sudden hearing loss] patients, compared to age- and sex-matched controls[,] . . . the difference between patients and controls did not remain significant in multivariate analysis." Pet. Ex. 92 at 5; see also Resp. Ex. F at 1. Moreover, Rudack et al. concluded that their data "suggested that fibrinogen is a marker for increased [sudden hearing loss] risk. However, that data does not support a role of fibrinogen as an independent risk factor for [sudden hearing loss]." Pet. Ex. 92 at 5.

Further, Dr. Ying noted that Weiss et al. studied fibrinogen after "idiopathic sudden hearing loss," not after flu vaccination. Resp. Ex. F at 1 (citing Pet. Ex. 103).

Dr. Ying's sixth criticism was Dr. Monsell's failure to explain the acute phase response specific to the ear which could cause hearing loss. Resp. Ex. F at 1-2. She noted the Liuba et al. findings were "not ear organ specific." Id. at 2 (emphasis omitted) (citing Pet. Ex. 36 at 102).

Her seventh reason was that Dr. Monsell's mechanism was so broad that "any stimuli or change in condition could potentially trigger the inflammatory response described by Dr. Monsell." Resp. Ex. F at 2. By way of example, she referenced Dr. Monsell's statement, regarding Neng et al., that "[a]ctivated pericytes . . . break down the blood-tissue barrier, allowing other potential pathogens or toxins to enter the cochlear tissue." Id. (quoting Pet. Ex. 63 at 8 (citing Pet. Ex. 85 at 6 fig.4)). According to Dr. Ying, this statement suggested that "other pathogens/toxins, unrelated to the [flu] vaccine, can enter the cochlea[] causing inner ear injury resulting in SSNHL." Id.

Moreover, Dr. Ying explained SSNHL has over one hundred potential etiologies, including infectious, otologic, traumatic, vascular, hematologic, neoplastic, and other causes. Resp. Ex. C at 4, 7 (citing Resp. Ex. C.4 at 1, 3).[90] And a cause is identified in seven to 45% of patients. Id. at 4. Most patients with SSNHL do not have an identifiable cause and are classified as idiopathic. Id. at 4, 7.

[90] Justin K. Chau et al., Systematic Review of the Evidence for the Etiology of Adult Sudden Sensorineural Hearing Loss, 120 Laryngoscope 1011 (2010). This article was also cited as Resp. Ex. F.1.

Lastly, regarding case reports, Dr. Ying asserted "case reports provide, at most, evidence of a temporal association between vaccines and adverse events, and not vaccine causation." Resp. Ex. C at 7.

In conclusion, Dr. Ying opined Dr. Monsell failed to provide evidence of "[flu] vaccine-induced inflammation causing SSNHL." Resp. Ex. F at 2. Petitioner's proposed inflammatory mechanism is "a general theory of 'idiosyncratic micro-vascular and inflammatory reactions' without evidence in the literature . . . specific and supportive for [flu] vaccine caused SSNHL." Id. at 4.

**ii. Althen Prong Two**

Dr. Ying opined that Petitioner's assertion that the flu vaccination was the cause of his hearing loss and tinnitus was not based on "a reasonable degree of medical probability." Resp. Ex. C at 7.

Dr. Ying opined Petitioner's SSNHL was, more like than not, idiopathic. Resp. Ex. F at 5. Although a defined cause is identified in 7-45% of patients, the majority of patients have no identifiable cause for their hearing loss, and thus, their hearing loss is "idiopathic." Id. at 2.

Next, Dr. Ying disagreed that Dr. Monsell's theory is applicable to Petitioner's case. She explained that Petitioner received high-dose steroids within five days of his hearing loss and did not experience improvement. Resp. Ex. C at 5. Merchant et al., cited by Dr. Monsell, noted that steroids are "potent inhibitors" of NFKB. Id. (quoting Pet. Ex. 40 at 10). Because Petitioner did not improve with steroids, reliance on the NFKB system is not supported, and thus, Dr. Ying opined this evidence weighs against Dr. Monsell's theory. Id.

Another reason Dr. Ying disagreed with Dr. Monsell's theory is based on the fact that Petitioner's hearing loss was unilateral. Resp. Ex. C at 6. Dr. Monsell used the example of autoimmune hearing loss (AIED) as support for his position that an inflammatory process can cause hearing loss; however, Dr. Monsell noted that typically, AIED progressively affects hearing loss in "both ears over weeks to months." Id. (quoting Pet. Ex. 16 at 10-11). As noted by Dr. Ying, Petitioner did not have bilateral hearing loss. Id. Dr. Ying noted that the case reported by Kolarov et al. involved a patient with normal hearing who developed bilateral hearing loss two days after receiving the flu vaccine. Id. at 7 (citing Pet. Ex. 34 at 1). Further, the patient received Xanaflu, a vaccine that contained trace amounts of gentamicin, which is known for its ototoxicity, whereas the flu vaccine given to Petitioner did not contain ototoxic components. Id.

Moreover, Petitioner received flu vaccinations for at least the two years prior to the vaccine in question, and he did not have any reported hypersensitivity, allergic reaction, or other adverse reaction. Resp. Ex. C at 7.

Lastly, Dr. Ying proposed that Petitioner's hyperlipidemia was "a recognizable factor" for SSNHL. Resp. Ex. F at 3. While she did not propose "[Petitioner's] SSNHL was definitely caused by hyperlipidemia," she noted that Petitioner had discontinued Zocor, which he was

taking for his hyperlipidemia, and this discontinuation may have compromised the microcirculation in his ear, which could have led to SSNHL. Id. Dr. Ying agreed with Dr. Monsell that risk factors were not the same as causal mechanisms, but she explained that risk factors can "provide a differential diagnosis with its own pathophysiology." Id. (quoting Pet. Ex. 63 at 17). Moreover, Petitioner's slight improvement in hearing observed in his audiogram on November 30, 2015 "suggests a possible transient total hearing compromise with limited hearing recovery that could be related to the inner ear circulation." Resp. Ex. C at 6. Therefore, Dr. Ying opined an acute vascular occlusion could not be ruled out as the cause of Petitioner's hearing loss. Id. (citing Pet. Ex. 52 at 4).

#### iii. Althen Prong Three

Dr. Ying does not dispute Petitioner developed hearing loss two days post-vaccination; however, she opined that a "temporal relationship" associating the flu vaccination to Petitioner's hearing loss "does not translate to causality for his developing SSNHL." Resp. Ex. F at 4; see also Resp. Ex. C at 1.

## IV. DISCUSSION

### A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors

unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

### B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such

testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not rigidly bound by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (noting Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

### C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d. 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether a petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and

noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

## V. CAUSATION ANALYSIS

### A. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds Petitioner failed to provide preponderant evidence of a sound and reliable theory to explain how the flu vaccines can cause SSNHL. There are several reasons for this finding.

At the outset, the undersigned notes that the pathogenesis of SSNHL is not known. The literature cited by the experts is consistent on this point. See, e.g., Resp. Ex. C.4 at 2 ("The causes of SSNHL are speculative and probably multifactorial . . . ."); Pet. Ex. 40 at 1 ("The cause and pathogenesis of idiopathic [SSNHL] remain unknown."); Pet. Ex. 69 at 1 ("[T]he pathogenesis is still largely unknown even today and is subject to controversial discussion.").[91]

Suggested causes of SSNHL are numerous and include infections, vascular insult, and inflammatory processes, among others. For each possible etiology, a different pathophysiological process is described. And there are inconsistencies between studies and papers about these posited etiologies. See, e.g., Pet. Ex. 35 at 1 ("Analysis of human temporal

[91] Martin Canis et al., Fibrinogen/LDL Apheresis Is a Promising Rescue Therapy for Sudden Sensorineural Hearing Loss, 7 Clinical Rsch. Cardiology Supplements 36 (2012).

bones from patients with [] [SSNHL] does not support a vascular insufficiency but is more suggestive of a viral etiology."). When the cause is vascular insufficiency, "it is usually in conjunction with some known systemic pathological process such as leukemia or intracranial lesions . . . or surgical interventions." Id. at 3-4. SSNHL has also been linked to risk factors such as diabetes and hypertension, but "findings are not consistent from study to study." Id. at 4.

Dr. Monsell's seven-step theory is an amalgamation of all the theories discussed in the numerous medical articles. When stacked on top of each other, the result is not a sound and reliable causal mechanism. Instead, it is a confusing puzzle. Pieces of the puzzle are not explained, foundational evidence is lacking, or the evidence is not relevant because the underlying facts and circumstances are different.

Moreover, Dr. Monsell did not provide evidence to support step one—that the flu vaccine stimulates the Acute Phase Reaction, or APR—of his seven-step mechanism. Since the six other steps of the seven-step theory rely on the first step, the entire mechanism fails.

For example, the first step of Dr. Monsell's theory asserts that the flu vaccine stimulates the APR, in which key proteins are released which may lead to a potentially undesired effect. He relies on Khalil and Al-Humadi who identify proteins identified after flu vaccination, including CRP and transthyretin, and they note that CRP is a diagnostic marker for inflammation. But the authors did not attribute an adverse reaction to CRP. Other examples of how the medical literature does not support the steps posited by Dr. Monsell are provided above, in the undersigned's review and analysis of the medical literature.

Further, Respondent's experts, Dr. Staats and Dr. Ying, describe how and why the medical literature and medical records are insufficient to show that the flu vaccine can cause SSNHL. The undersigned finds their explanations more consistent with the literature, and therefore, their opinions are more persuasive.

Respondent's experts explain that some of the studies Dr. Monsell relies upon were performed using methodology that is not comparable to vaccination, and thus, it is not reasonable to extrapolate the findings to vaccination. As explained by Dr. Staats, "LPS-induced inflammation is not a model that is relevant to vaccine-induced inflammation because the magnitude and duration of LPS-induced inflammation is much greater than that inflammation induced by [flu] vaccines." Resp. Ex. E at 4. Dr. Ying agreed, noting that "LPS-induced inflammation is not a model that is applicable to vaccine-induced inflammation." Resp. Ex. F at 4. The undersigned agrees with Respondent's experts on this point, as their reasoning is more factually sound and persuasive.

The same criticism applies to Petitioner's reliance on inferences that a live virus flu infection causes the same inflammatory response as the inactivated flu vaccine. Respondent's experts were persuasive in showing why studies applicable to the flu infection cannot be extrapolated to an inactivated vaccine. "An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of

Health & Hum. Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280). Here, Dr. Monsell failed to explain how data from the live flu virus could be extrapolated to the inactivated flu vaccine at issue here.

Petitioner need not make a specific type of evidentiary showing or require identification of a specific antigenic trigger for an immune-mediated pathology to prove that a theory is sound and reliable by preponderant evidence. Given the state of current scientific knowledge, there is no way that a petitioner could satisfy such a requirement. Requiring proof of the identify of a specific antigen to prove causation would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program").

However, based on the current understanding of SSNHL as described in the literature filed herein, and as explained by Respondent's experts, Dr. Monsell's proposed mechanism falls short of sound and reliable.

Overall, the undersigned finds that here, Petitioner's seven-step theory in part or as a whole is unsupported by medical or scientific facts, research, or any other reliable evidence. Moreover, his theories are speculative and/or conclusory in nature. When evaluating whether petitioners have carried their burden of proof, special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. See id.

Next, Dr. Monsell used the word "possibility" in several places in his expert report. For example, prior to introducing his multi-step mechanism, Dr. Monsell wrote "[flu] vaccination may increase the possibility of SSNHL directly or indirectly triggered by any one or any combination of the [listed] causal and/or conditions events." Pet. Ex. 63 at 6. When referencing the NFKB pathway and induction of cytokines, Dr. Monsell wrote that the studies he cited "demonstrate the possibility that [the flu] vaccination could activate the NFKB system in the inner ear." Pet. Ex. 16 at 15; see also Pet. Ex. 63 at 4 ("The findings that these cells may be selectively stressed by systemic inflammation raises the possibility that their vulnerability systemically administered inflammatory stress may underlie two poorly understood forms of hearing loss, sudden hearing loss and immune-mediated hearing loss."). Dr. Monsell also concluded "[t]he studies cited by [] [R]espondent provide no sound basis to exclude the possibility of SSNHL due to [the flu] vaccination." Pet. Ex. 63 at 21. Opinions expressed as "possibilities" are not consistent with the burden of proof or preponderant evidence, as required in this case.

Opinions expressed as possibilities are not sufficient to establish causation. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence).

Lastly, Vaccine Program case law does not support causation here. In Vanore, a case where compensation was denied, Petitioner's expert was Dr. Monsell and he offered the same seven-step theory to explain how the flu vaccine could cause SSHNL. Vanore v. Sec'y of Health & Hum. Servs., No. 21-0870V, 2024 WL 3200287 (Fed. Cl. Spec. Mstr. May 31, 2024). In Vanore, the Chief Special Master analyzed Dr. Monsell's seven-step theory and found Petitioner was not entitled to compensation, in part, because Dr. Monsell's theory was unreliable and not persuasive. Id. at *19-20.

Moreover, there are other flu vaccine and hearing loss cases where compensation has been denied.[92] See, e.g., M.R. v. Sec'y of Health & Hum. Servs., No. 16-1024V, 2023 WL 4936727 (Fed. Cl. Spec. Mstr. June 30, 2023) (determining the petitioner's acoustic neuroma/vestibular schwannoma, not the flu vaccine, was the most likely cause of his SSNHL); Donica v. Sec'y of Health & Hum. Servs., No. 08-625V, 2010 WL 3735707, at *1, 10 (Fed. Cl. Spec. Mstr. Aug. 31, 2010) (finding flu vaccine was not demonstrated to cause adult hearing loss in adult but not making a determination as to Althen prong one because prong three was not met).

Petitioner in Inamdar alleged the flu vaccine caused his SNHL. Inamdar v. Sec'y of Health & Hum. Servs., No. 15-1173V, 2019 WL 1160341, at *1 (Fed. Cl. Spec. Mstr. Feb. 8, 2019). Petitioner proposed the flu vaccine "could cause the production of proinflammatory cytokines immediately upon vaccine administration," and alternatively, that specific components of the vaccine "were structurally homologous with ganglioside receptors on the neuronal myelin contained in the inner ear tissue, and that antibodies generated in response to the vaccine could also cross-react with the self-myelin, resulting in tissue damage." Id. at *5-6. The Chief Special Master found the first theory relied too heavily on what was known about the wild virus rather than the vaccine, and further found that both theories were unsupported by the literature. Id. at *17-18. An alternative cause also existed, and a one-day onset was not shown to be medically acceptable. Id. at *18-19.

In Kelly, the Petitioner alleged the flu vaccine caused his SNHL. Kelly v. Sec'y of Health & Hum. Servs., No. 16-878V, 2021 WL 5276373, at *1 (Fed. Cl. Spec. Mstr. Oct. 18, 2021), mot. for rev. den'd, 160 Fed. Cl. 316 (2022). Petitioner alleged a significant aggravation claim, but the Chief Special Master noted his determination would have been the same even if Petitioner alleged a causation-in-fact claim. Id. at *24. Petitioner proposed a Type I sensitivity reaction and alternatively, an autoimmune response. Id. at *25-26. The Chief Special Master

[92] In addition to the flu vaccine, cases involving other vaccines have also found against compensation. See, e.g., Hopkins v. Sec'y of Health & Hum. Servs., No. 00-745V, 2007 WL 2454038, at *1, *19-23 (Fed. Cl. Spec. Mstr. Aug. 10, 2007) (dismissing claim of SNHL due to *haemophilus influenzae* type B, diphtheria-tetanus-pertussis, and oral polio vaccines and finding the submitted literature failed to support the theory), aff'd, 84 Fed. Cl. 517 (2008).

found limited support for the primary theory and found the autoimmune theory inconsistent with the facts presented, including the fact that Petitioner's hearing loss was unilateral, and the onset was two hours. Id. at *24-26. It was also noted that "an autoimmune origin would in most cases mean some other underlying systemic disease was occurring" and there was nothing in the record to support that. Id. at *26 (emphasis omitted).

The undersigned has also denied entitlement in two hearing loss cases. In Alsaadeh, the undersigned denied entitlement where Petitioner alleged the flu and Prevnar 13 vaccines caused his hearing loss. Alsaadeh v. Sec'y of Health & Hum. Servs., No. 19-1097V, 2024 WL 694072 (Fed. Cl. Spec. Mstr. Jan. 23, 2024). The undersigned found Petitioner's immune-mediated theory was not sound or reliable in part because the causal mechanism of immune-mediated hearing loss is unknown. Id. at *31-34. In Herms, the undersigned also denied entitlement where petitioner alleged that the diphtheria tetanus toxoid acellular pertussis vaccination that she received caused SNHL and tinnitus in her left ear. Herms v. Sec'y of Health & Hum. Servs., No. 19-70V, 2024 WL 1340669, at *1 (Fed. Cl. Spec. Mstr. Mar. 4, 2024), mot. for rev. den'd, aff'd, 173 Fed. Cl. 1, appeal docketed, No. 25-1007 (Fed. Cir. Sept. 27, 2024). The undersigned found Petitioner's theory was not sound or reliable because it was underdeveloped, conclusory in nature, and vague and because the causal mechanism of immune-mediated hearing loss is unknown. Id. at *19-20.

Although decisions of other special masters are not binding, the undersigned generally agrees with the reasoning of her colleagues in the above cases. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

While there is one reasoned decision where entitlement was granted to a petitioner who alleged the flu vaccine caused SSNHL, the undersigned notes that the facts and theory are different. Madigan v. Sec'y of Health & Hum. Servs., No. 14-1187V, 2021 WL 3046614 (Fed. Cl. Spec. Mstr. June 25, 2021).

Overall, the majority of Vaccine Program cases have resulted in denial in compensation to petitioners. This outcome largely tracks the medical literature, which upon review, illustrates that the cause of hearing loss is unknown. The lack of knowledge cannot be supplanted by supposition.

In summary, Petitioner has failed to offer a sound and reliable medical theory in support of his claim. Thus, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to the first Althen prong.

### B. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause-and-effect showing the October 23, 2018 flu vaccine to be the cause of Petitioner's SSNHL. See Althen, 418 F.3d at 1278. There as three reasons for this finding.

First, the records and expert opinions show aspects of Petitioner's clinical course are not consistent with the mechanism posited by Dr. Monsell. To start, Petitioner received high-dose steroids, which would be expected to improve his hearing if the etiology was inflammatory in nature. Further, steroids are strong inhibitors of the NFKB pathway. Steroids did not improve Petitioner's hearing loss, which weighs against Dr. Monsell's theory.

Additionally, Petitioner's hearing loss was unilateral and acute. Autoimmune hearing loss typically affects both ears in a progressive course. Additionally, in the case reported by Kolarov et al., the individual experienced bilateral hearing loss after a flu vaccination that contained different ingredients than Petitioner's flu vaccine.

The second reason is based on a potential alternative explanation for Petitioner's hearing loss. Dr. Ying explained that Petitioner had a known risk factor for hearing loss, hyperlipidemia, for which he took Zocor. He had discontinued his Zocor prior to his hearing loss which may have affected his microcirculation in the ear and could have led to his hearing loss. While Dr. Ying agreed with Dr. Monsell that risk factors differed from causal mechanisms, she explained that risk factors can provide a differential diagnosis supported by pathophysiology. Although Dr. Ying did not state that Petitioner's hyperlipidemia combined with a discontinuation of the medication treating that condition more likely than not caused Petitioner's hearing loss, the combination of these two factors present a possible alternative cause—an acute vascular occlusion as the cause of Petitioner's hearing loss.

The undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding petitioner does not bear the burden of eliminating alternative independent potential causes). However, she finds it reasonable to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in

question." Stone, 676 F.3d at 1379; see also Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 963 (Fed. Cir. 2023) ("Such contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law."); Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case.").

Third, Petitioner's physicians did not attribute his hearing loss to vaccination. The first health care provider who saw and treated Petitioner was NP Kastner three days after vaccination, on October 26, 2018. She noted that Petitioner had associated allergy symptoms and that his symptoms might be due to fluid in his middle and inner ear due to his chronic allergies. Her assessment was obstruction of right eustachian tube.

Shortly thereafter, Petitioner saw a specialist, otolaryngologist Dr. Holm, who noted Petitioner had a history of noise exposure, although he used ear protection. Subsequently, Petitioner called Dr. Holm to ask whether the flu shot he received a couple of days before onset could have caused his hearing loss. The records show that Dr. Holm advised that "the flu shot would not cause current symptoms." Pet. Ex. 3 at 161.

One year later, in October 2019, Petitioner's PCP, Dr. Zuzick wrote that Petitioner had a history of hearing loss "which may be related to receiving a [flu] vaccination." Pet. Ex. 12 at 36. In February 2020, Dr. Zuzick's records reflect a phone call where Petitioner reported that he discussed his hearing loss with Dr. Zuzick at the prior visit. The note states "Petitioner had testing done and none of the doctors attributed his hearing loss to the flu shot but the patient does." Id. at 45. When read and interpreted together, these two notes suggest that Petitioner reported his hearing loss to Dr. Zuzick, as well as his belief that his hearing loss was due to vaccination, and not that Dr. Zuzick believed that Petitioner's hearing loss was vaccine related. In the alternative, if the first note in 2019 is interpreted as Dr. Zuzick's opinion that Petitioner's hearing loss was related to his flu vaccination, the undersigned finds it carries less weight than the records of the specialist in hearing loss, otolaryngologist Dr. Holm.

In summary, Petitioner was seen by several health care providers. Of these, specialist Dr. Holm specifically opined that the flu shot would not cause Petitioner's current symptoms of hearing loss.

Generally, treating physician statements are typically "favored" as treating physicians "are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280). "In weighing the persuasiveness of opinion testimony, special masters may consider the relative expertise of the witness." Koehn v. Sec'y of Health & Hum. Servs., No. 11-355V, 2013 WL 3214877, at *32 (Fed. Cl. Spec. Mstr. May 30, 2013), aff'd, 773 F.3d 1239 (Fed. Cir. 2014); see also Dwyer v. Sec'y of Health & Hum. Servs., No. 03-1202V, 2010 WL 892250, at *64 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (giving greater weight to M.D. epidemiologists' opinions on medical issues than to Ph.D. epidemiologist's opinion); Pafford, 451 F.3d at 1359 (affirming the special master's rejection of expert's testimony because he lacked proper qualifications in the specialty areas in which he testified).

Lastly, with regard to Petitioner's argument that the VAERS report provides evidence of causation, the undersigned disagrees. The VAERS report simply provides an overview of the circumstances. Further, it was authored by a pharmacist, and the pharmacist did not offer a causal opinion.

Accordingly, the undersigned finds that Petitioner has not satisfied his burden under Althen prong two.

### C. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also be consistent with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn, 773 F.3d at 1243; Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358.

Petitioner received a flu vaccine on October 23, 2018. Two days later, on October 25, 2018, Petitioner experienced right-sided hearing loss, later diagnosed as SSNHL. The experts agree Petitioner's onset was two days post-vaccination. Respondent's experts did not disagree that there was a temporal association between vaccination and the onset of hearing loss. However, they agree that a temporal relationship without other evidence is insufficient to support causation.

A temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Thus, even though Petitioner has provided preponderant evidence satisfying Althen prong three, Petitioner is not entitled to compensation.

## VI. CONCLUSION

The undersigned extends her sympathy to Petitioner for all that he has suffered due to his hearing loss. Her Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination he received caused him to develop SSNHL. Therefore, Petitioner is not entitled to compensation and the petition must be dismissed.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

**IT IS SO ORDERED.**

**s/Nora Beth Dorsey**
Nora Beth Dorsey
Special Master